

Dissertation on

COMPARATIVE STUDY FOR THE PURPOSE OF SAFETY
AND EFFICACY OF 0.5% ROPIVACAINE WITHOUT
EPINEPHRINE, 0.5% ROPIVACAINE WITH EPINEPHRINE
AND 0.5% BUPIVACAINE IN PATIENTS RECEIVING
SUBCLAVIAN PERIVASCULAR BRACHIAL PLEXUS BLOCK

Submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

*In partial fulfillment of the requirements
for the award of degree of*

**MD (BRANCH - X)
ANAESTHESIOLOGY**



**GOVERNMENT STANLEY MEDICAL
COLLEGE & HOSPITAL**

**THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY
CHENNAI, TAMILNADU**

APRIL- 2011

CERTIFICATE

This is to certify that this dissertation entitled “COMPARATIVE STUDY FOR THE PURPOSE OF SAFETY AND EFFICACY OF 0.5% ROPIVACAINE WITHOUT EPINEPHRINE, 0.5% ROPIVACAINE WITH EPINEPHRINE AND 0.5% BUPIVACAINE IN PATIENTS RECEIVING SUBCLAVIAN PERIVASCULAR BRACHIAL PLEXUS BLOCK” is bonafide record work done by **Dr.K.SATHISHAKUMAR** under the guidance of Prof. Dr.Ponnambala Namachivayam, Additional Professor, Department of Anaesthesiology, submitted to the Tamil Nadu Dr. M.G.R. Medical University in partial fulfillment of University regulation for MD, Branch X –Anaesthesiology.

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DECLARATION

I **Dr.K.SATHISHAKUMAR** solemnly declare that this dissertation titled “COMPARATIVE STUDY FOR THE PURPOSE OF SAFETY AND EFFICACY OF 0.5% ROPIVACAINE WITHOUT EPINEPHRINE, 0.5% ROPIVACAINE WITH EPINEPHRINE AND 0.5% BUPIVACAINE IN PATIENTS RECEIVING SUBCLAVIAN PERIVASCULAR BRACHIAL PLEXUS BLOCK” has been done by me. I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, diploma to any other University board either in India or abroad.

This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfillment of the rules and regulation for the award of Doctor of Medicine degree Branch –X (Anaesthesiology) to be held in APRIL 2011.

Dr. K.Sathishakumar

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Introduction

INTRODUCTION

Peripheral nerve blocks are gaining widespread popularity for perioperative pain management because of their distinct advantages over general and central neuraxial anesthesia. Pain relief with peripheral nerve block (PNB) is devoid of side effects such as somnolence, nausea, vomiting, hemodynamic instability and voiding difficulties inherent to general and central neuraxial anesthesia.

Brachial plexus blocks (interscalene, supraclavicular and axillary blocks) ¹⁻¹⁰ provide a useful alternative to general anesthesia for upper limb surgery. Each of these routes has its own particular advantages, disadvantages and complications. They achieve ideal operating conditions by producing complete muscular relaxation, maintaining stable intraoperative hemodynamic, and the associated sympathetic block. The sympathetic block decreases postoperative pain, vasospasm and edema. Supraclavicular perivascular brachial plexus block is a very commonly practised technique in patients undergoing surgery on forearm and hand. Supraclavicular block as opposed to infraclavicular brachial plexus block generally offers denser and complete upper limb anaesthesia. Nerve stimulators are now widely seen as useful aids in nerve

blocks ^(11,12). Its use avoids paraesthesia, decreases the chance of nerve injury and gives high success rate.

Introduction of long acting local anesthetic with better safety profile as well as better equipment has further increased the usage of peripheral nerve blocks. Bupivacaine hydrochloride, a long acting local anaesthetic agent, has been used extensively for supraclavicular brachial plexus block ¹³. Ropivacaine ¹⁴⁻²⁶, the S- enantiomer of S-1-propyl-2,6-pipecoloxylidide, is an amino-amide local anaesthetic with chemical structure similar to that of bupivacaine. Ropivacaine is a potent blocker of A delta and C fibres (pain fibres). It has been reported to be less toxic than Bupivacaine.

In contrast to Bupivacaine which is a racemic mixture of R and S enantiomers, Ropivacaine is the first local anesthetic that has developed as a pure enantiomer. S enantiomer is considered less neurotoxic and cardiotoxic ⁴² than the R enantiomer of local anesthetics, perhaps reflecting different pharmacodynamics, pharmacokinetics and toxicities. Studies ^{18,23-26} have shown that Ropivacaine and Bupivacaine when given in equal volumes was similar in terms of onset and duration of sensory block and frequency of motor block.

The addition of epinephrine does not prolong the duration of Ropivacaine in subclavian perivascular brachial plexus²⁷⁻²⁸ or epidural block. Low concentrations of Ropivacaine may produce clinically significant vasoconstriction which is not further increased by the addition of epinephrine.

This study attempts to compare the safety and efficacy of 0.5% Ropivacaine without epinephrine and 0.5% Ropivacaine with epinephrine and 0.5% Bupivacaine in patients receiving subclavian perivascular brachial plexus block.

Aim of the Study

AIM OF THE STUDY

To study the efficacy and safety of 0.5% Ropivacaine without epinephrine and 0.5% Ropivacaine with epinephrine and 0.5% Bupivacaine in patients receiving subclavian perivascular brachial plexus block.

History

HISTORY

The first effort for the achievement of local or regional anesthesia dates back to the middle ages, where Ambroise Pare described in 1564 in France “that anesthesia was used to carry out operations at the extremities”, with the help of the compression mechanism intended to bring about analgesia, while in 1846 in Italy M.A. Severino brings about local anesthesia by putting ice or snow on the surgical field²⁹. Nevertheless, the real regional anesthesia started upon discovery of the holed metallic wire in 1854 by Wood, as well as the local anesthetic properties of cocaine²⁹.

Cocaine is contained in the leaves of a plant, the mahogany of coca, which is grown in the South American countries^{29,30}. Cocaine was isolated from the leaves of the coca's mahogany for the first time in 1860, by A. Niemann in Germany. The substance was crystalline, colourless, odourless and with a sour taste. In 1884 Karl Koller from Vienna described at the conference of German ophthalmologists in Haideberg the anesthetic qualities of a 2% cocaine solution after it was instilled in the eye³¹. In 1885 Halstead, on the one hand, gave a description of the exclusion of the nerve through infiltration with cocaine, and Corning from the United States, on the other hand, gave a description of the episcleroid anesthesia, which most probably, though, corresponded

to the paravertebral exclusion. Thus, cocaine, managed to acquire the prestige it deserved as a local anesthetic after a number of fatal implications was recorded from the acid action of the solutions with a high cocaine content that they were using at that time.

The revolution in the history of local anesthetics broke in 1904, when Einhorn introduced novocaine (procaine), a local anesthetic with fewer side effects than cocaine. Nevertheless, the duration of its action has been short-lived, a fact which has limited its use for the most part to operations with a short duration. This problem was solved by Braun, who proposed the addition of adrenaline to the local anesthetic, for the purpose of prolonging both the duration and the validity of the local anesthetic.

In 1943 Nils Lofgren composed lidocaine, which belongs to the group of aminoalcydamides . Beyond any doubt, the lidocaine has been the main substance and, at the same time, the base for all the later studies of the local anesthetics that followed. The later researches led to the discovery of new local anesthetics such as etidocaine, the prilocaine, and the bupivacaine.

THE DEVELOPMENT OF THE MODERN LOCAL ANESTHETICS

Albright's announcement on the quest of new local anesthetics in 1979 with regard to the heart failures following the administration of etidocaine and bupivacaine is believed to be significant ³². After this

announcement, there followed a constructive discussion with regard to the toxicity of the local anesthetics^{33, 34}. There followed experimental studies, which confirmed the assumption of Albright that deaths were the result of the high cardiotoxicity of the long-lasting local anesthetics³⁵⁻³⁸.

During the search for new local anesthetics, the researchers utilized the characteristic fabrication of the 3-dimensional molecular structure that the bupivacaine can demonstrate³⁹. The meaning of “stereo-isomery” had been introduced at the end of the 19th century by the Swede chemist von Berzelius (1779 through 1848). Thus, the Bupivacaine (racemic mixture with R- and S-form in the same percentage) helped produce ropivacaine (net S-enantiomer).

Ropivacaine is produced through the halcyliosis of the S-enantiomer of L-staphylytic-dibenzoic-acid-ester. Its chemical structure has a great similarity with that of bupivacaine and of mepivacaine, while its two-dimensional form has a few only differences from that of bupivacaine. Ropivacaine does not differ as regards the physical and chemical properties from racemic mixture. The only difference that it presents as compared with bupivacaine is its lipophilia, which is comparable with that of mepivacaine.

The significant progress that the introduction of this short-lived local anesthetic in the clinical practice relies, on the one hand, upon the

reduced toxicity on the cardiovascular and central nervous system and, on the other hand, on its dose-dependent differential closeout (the qualities of which we can utilize in the postoperative analgesia and the administration of epidural anesthesia in the obstetrics) ⁴⁰⁻⁴³. The wide acceptance of ropivacaine in the clinical practice was followed by the preparation of a number of other substances by the group of amides, for the purpose of introducing them into the clinical practice. Meanwhile, an S-enantiomer of bupivacaine, the levobupivacaine, which, based on the first clinical experiences, has proven the same profile of action, such as in the racemic mixture which has been used so far in the various techniques of the anesthesia area ^{44, 45}. Levobupivacaine is governed by an equally large lipophilia such as the bupivacaine and that for this reason it differs to a great extent from the ropivacaine, mainly as to the power, the kinetics on the receptor level and, quite possible, as to the cardiac toxicity. Due to the high lipophilia which governs it, the power of the levobupivacaine is higher than of ropivacaine, while its kinetics at the level of the receptor is quite probably comparable to that of Bupivacaine.

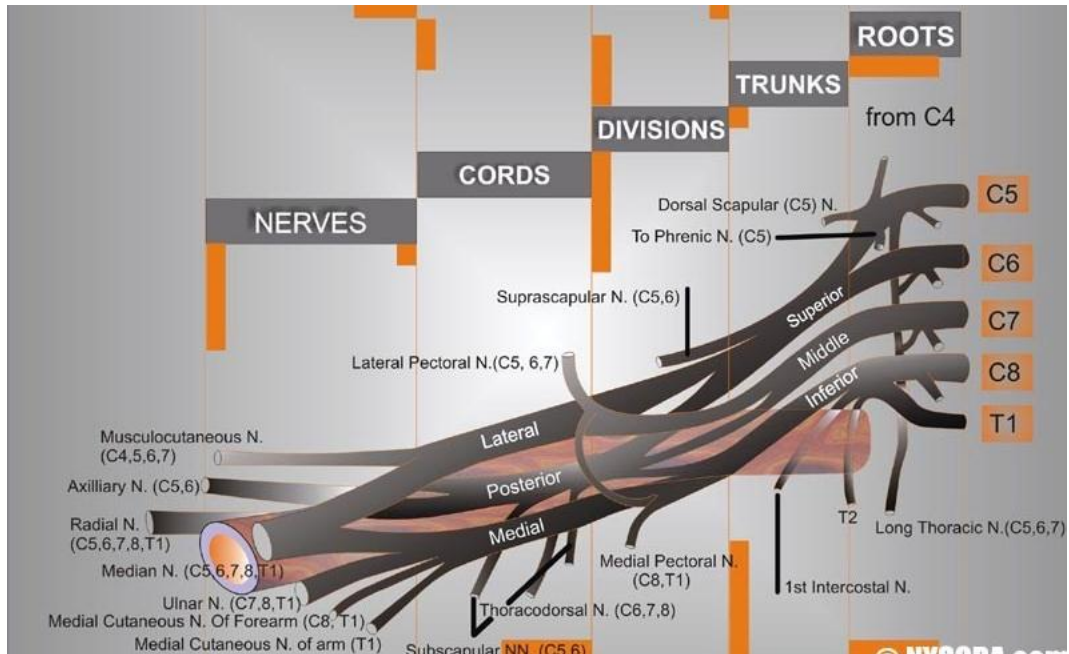
Anatomy of Brachial Plexus

ANATOMY OF BRACHIAL PLEXUS ⁴⁶⁻⁴⁹

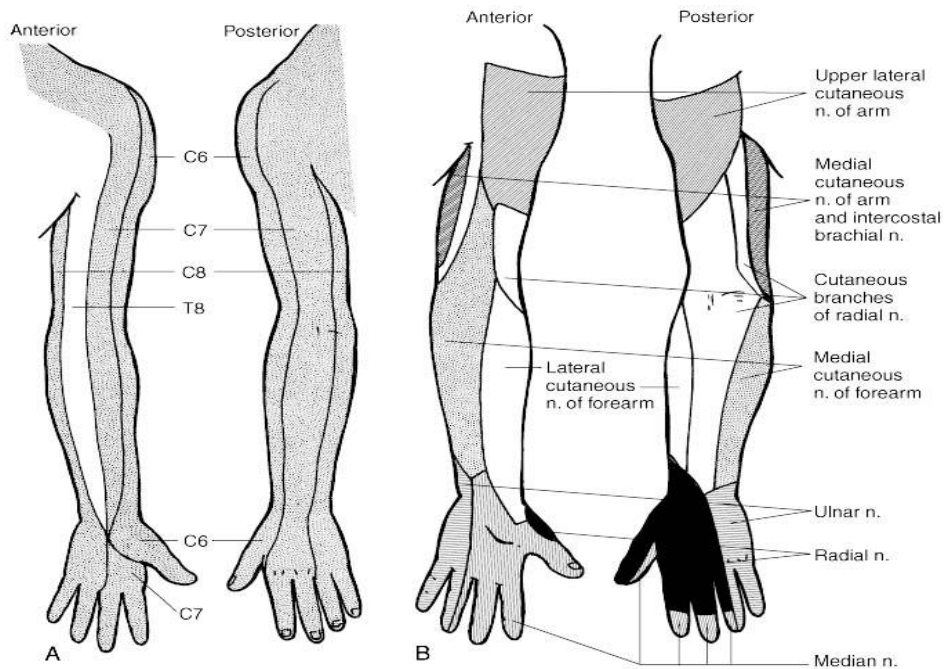
Knowledge of the formation of the brachial plexus and its distribution is essential to the intelligent and effective use of the brachial plexus blockade for the surgeries of the upper limb. Close familiarity with the vascular, muscular and fascial relationship of the plexus throughout the formation and distribution is equally essential to the mastery of various techniques of Brachial plexus Blockade.

In its course from the intervertebral foramina to the arm, the fibre's that constitute the plexus are composed consecutively of roots, trunks, divisions, cords and terminal branches, which are formed through a complex process of combining, dividing, recombining and finally redividing.

The brachial plexus is formed by the union of the anterior primary rami of the fifth, sixth, seventh, eighth cervical nerves and the first thoracic nerve, with variable contributions from the fourth cervical (pre fixed) above and second thoracic nerve (post fixed) below. These nerves unite to form trunks, which lie in the neck above the clavicle. Its roots pass between the scalenus anterior and scalenus medius which is enclosed by fascia accompanied by the subclavian artery and then invaginates the scalene fascia to form a neurovascular bundle. This fascia becomes the axillary sheath in the axilla.



ANATOMY OF BRACHIAL PLEXUS



SENSORY INNERVATIONS OR UPPER LIMB

RELATION OF BRACHIAL PLEXUS:**Anterior relations:**

The skin, superficial fascia, platysma, supraclavicular branches of the superficial cervical plexus, deep fascia and external jugular vein. The clavicle is in front of the lower part and scalenus anterior is in the front of the upper part.

Posterior relations:

Scalenus medius and the long thoracic nerve of bell.

Inferior relations:

Related to the first rib.

Superior relations:

Lies first above and then lateral to the subclavian artery.

SYMPATHETIC CONTRIBUTION TO THE PLEXUS:

Close to the emergence, the fifth and sixth cervical nerves, each receive a grey ramus from the middle cervical sympathetic ganglion. The seventh and eighth cervical nerves each receive a grey ramus from the inferior cervical ganglion.

ROOTS:

Anterior primary rami of C5, C6, C7, C8 and T1 (occasionally C4 or T2)

TRUNKS:

- Upper trunk - Anterior rami C5 and C6
- Middle trunk – Anterior ramus of C7
- Lower trunk – Anterior rami of C8 and T1

DIVISIONS:

Behind the clavicle each trunk divides into anterior and posterior divisions.

CORDS:

- Lateral cord – Anterior divisions of upper and middle trunks (C5-C7)
- Medial cord – Anterior divisions of lower trunk (C8-T1)
- Posterior cord – Posterior divisions of all the three trunks (C5-T1)

BRANCHES:**From roots:**

- Nerve to Serratus anterior (C5, C6, and C7)
- Muscular branches to longus cervicis (C5, C6, C7, and C8)
- Nerve to Scalene (C5, C6, C7 and C8)
- Nerve to Rhomboids(C5)
- A twig to Phrenic nerve (C5)

From trunk:

- Nerve to subclavius (C5-C6)
- Suprascapular nerve (C5-C6)

From cords:**Lateral cord:**

- Lateral root of median nerve (C5, C6 and C7)
- Lateral pectoral nerve (C5, C6 and C7)
- Musculocutaneous nerve (C5, C6 and C7)

Medial cord:

- Medial root of median nerve (C8, T1)
- Medial cutaneous nerve of arm (C8, T1)
- Medial cutaneous nerve of forearm (C8, T1)
- Medial pectoral nerve (C8, T1)
- Ulnar nerve (C8, T1)

Posterior cord:

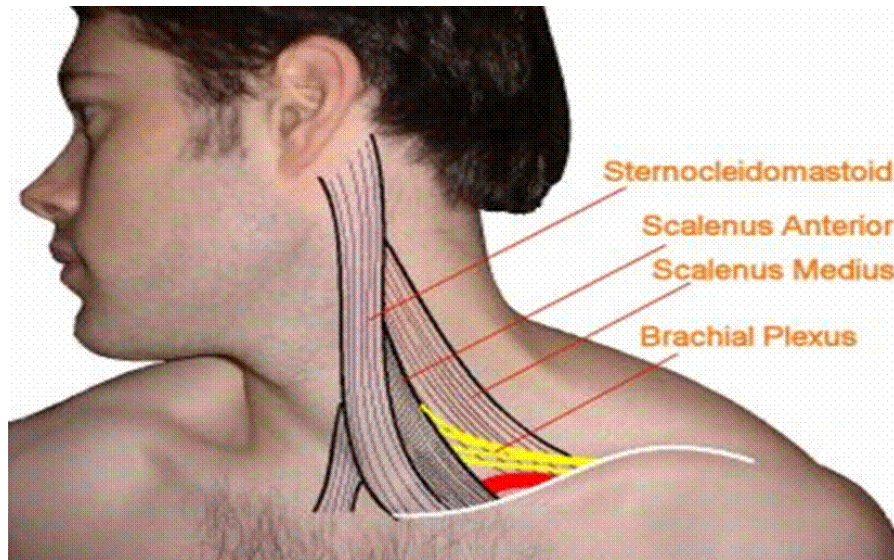
- Radial nerve (C5- T1)
- Axillary nerve (C5-C8)
- Upper and lower subscapular nerve (C5, C6)
- Nerve to latissimus dorsi (C6, C7, C8)

Familiarity with the perineural structures that surround and accompany the brachial plexus as it leaves the vertebral column on its course to the upper arm is as important as the knowledge of the formation

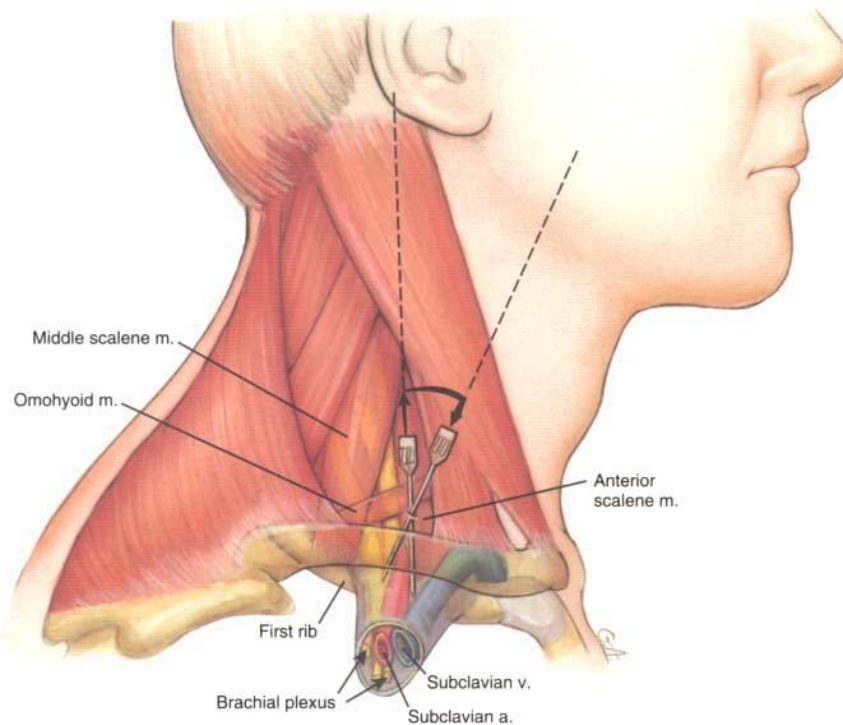
and distribution of the neural plexus itself. Palpable muscular and vascular landmarks allow accurate location of the plexus percutaneously. An appreciation of the fascial relations is absolutely essential since this is the basis for all the perivascular techniques.

After leaving the intervertebral foramina, the anterior primary rami of the nerves destined to become the brachial plexus travel in the gutter formed by the anterior and posterior tubercles of the corresponding transverse processes of the cervical vertebra. After leaving the transverse processes, the roots of the plexus descend in front of the middle scalene muscle, which arises from the posterior tubercles of the transverse processes of lower six cervical vertebra. The insertion of this muscle on the first rib is separated from that of the anterior scalene muscle by the inferior trunk of the brachial plexus. The anterior scalene muscle arises from the anterior tubercles of the transverse processes of the third to sixth cervical vertebrae and inserts on the scalene tubercle of the first rib, thus separating the subclavian artery from the subclavian vein.

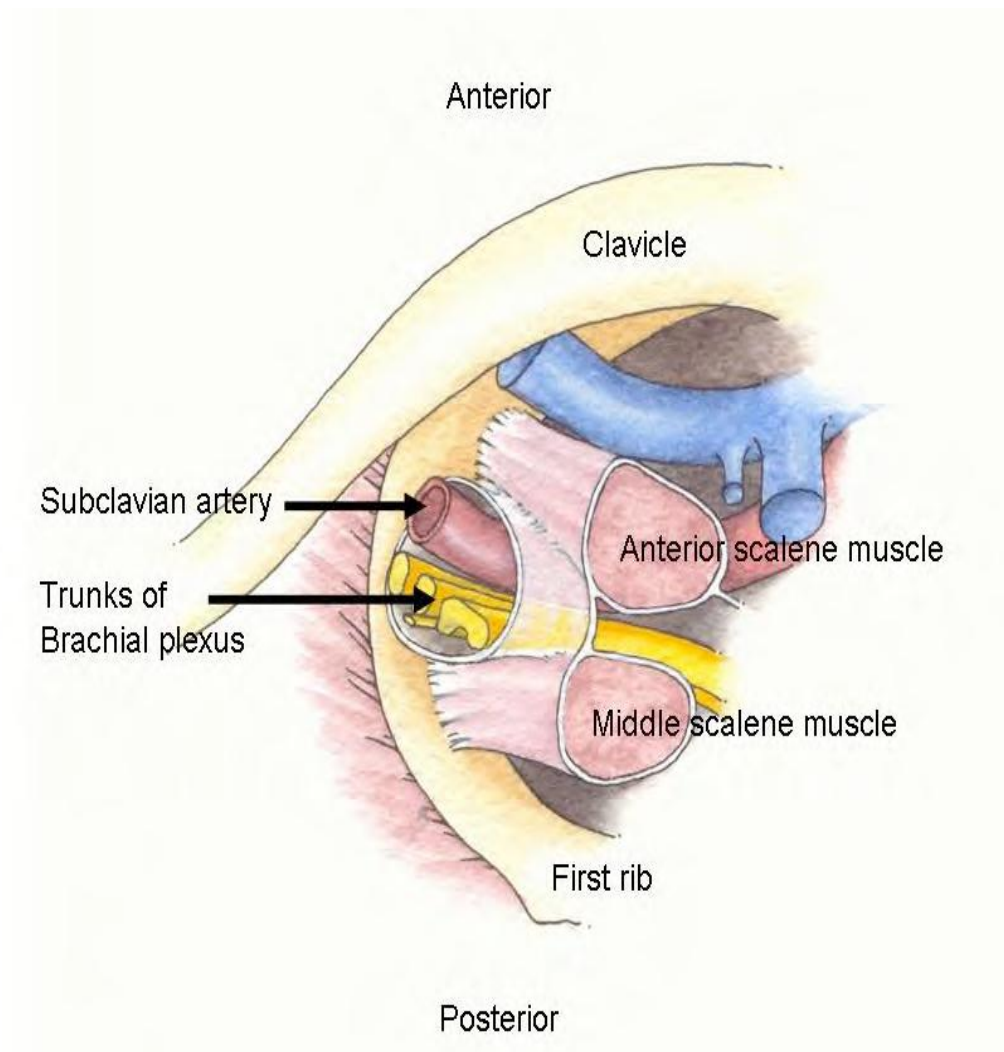
The fascia covering both the scalene muscle is derived from the prevertebral fascia, which splits to invest these muscles and then fuses again at their lateral margins to form an enclosed inter scalene space. Therefore as the roots leave the transverse processes, they emerge between the two walls of the fascia covering the anterior and middle scalene muscles. In their descend towards the first rib to form the trunks of the plexus the roots may be considered to be sandwiched between the anterior and middle scalene muscle, the fascia of which serves as a sheath



SURFACE ANATOMY FOR SUPRACLAVICULAR BLOCK



SUPRACLAVICULAR BLOCK: PERIVASCULAR AND PLUMB BOB APPROACHES



**SUBCLAVIAN PERIVASCULAR BLOCK ANATOMY: BRACHIAL PLEXUS
VIEWED FROM ABOVE**

of the plexus. As the trunks approach the first rib they are arranged one above the other vertically.

As the trunks of the plexus cross the first rib, they are joined by the subclavian artery, which lies in a plane anterior to the trunks, so that the inferior trunk lies behind the artery in the subclavian groove with the middle and superior trunks above the level of the vessel. At this level the artery and the trunks are moving laterally, across the ribs and invaginate the scalene fascia to form the subclavian perivascular space, which is continuous medially and superiorly with the interscalene space and inferiorly and laterally with the axillary perivascular space.

The important concept is that there is a continuous fascial enclosed perineural and perivascular space extending from the cervical transverse processes to several centimeters beyond the axilla; this space has been divided into a axillary perivascular space and interscalene space. The existence of such a continuous perineural space renders brachial plexus block simple. The space described may be entered at any level, and the volume of the anesthetic injected at this level would determine the extent of anesthesia. Thus, the technique to be used in any case should be determined on the basis of the surgical site, the required level of anesthesia, the physical status and habitus of the patient.

Brachial plexus can be blocked at the level of roots, trunks, cords, peripheral branches. The block at each level has a distinct distribution of anesthesia, advantages, disadvantages and complications.

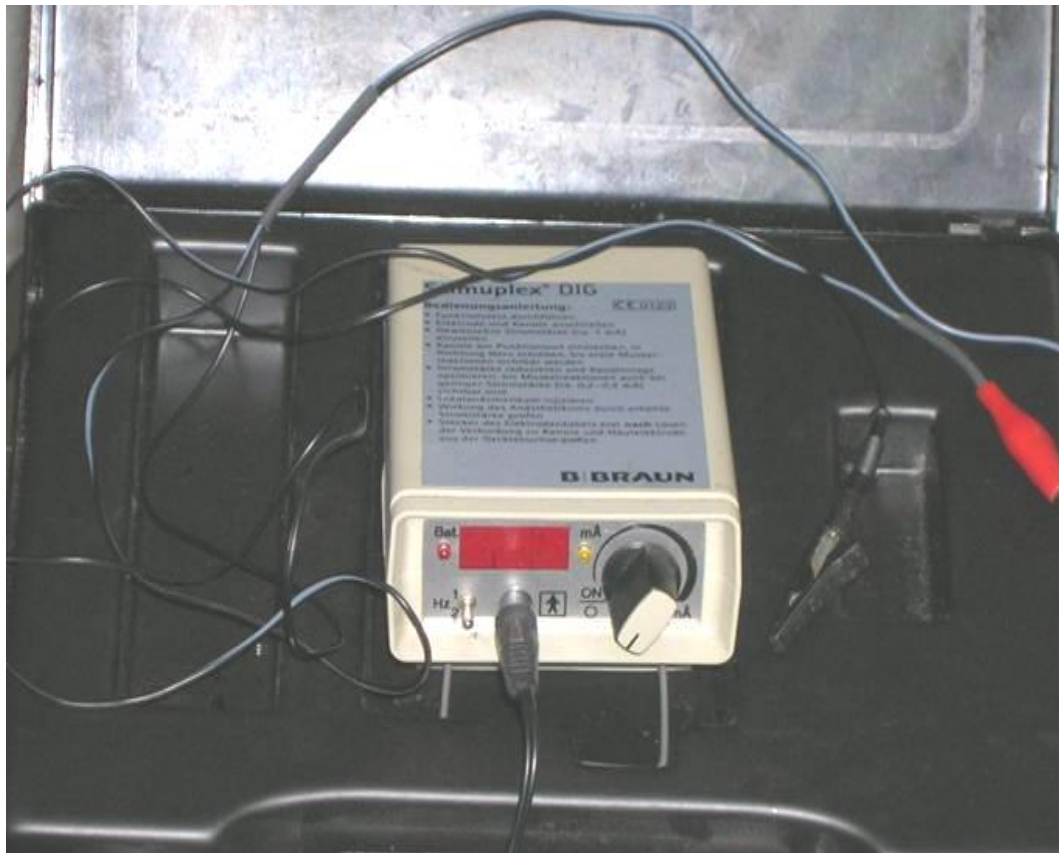
*Peripheral
Nerve
Stimulator
Technology*

PERIPHERAL NERVE STIMULATOR TECHNOLOGY^{11,12,50-56}

The ability of a nerve stimulator to evoke a motor response depends on the intensity, duration, and polarity of the stimulating current used and the needle (stimulus)-nerve distance. To propagate a nerve impulse, a threshold current must be applied to the nerve fibre. Peripheral nerve stimulation is typically performed using a rectangular pulse of current. When a square pulse of the current is used to stimulate a nerve, the total charge delivered is the product of the current strength and the duration of pulse.

RHEOBASE-is the minimal threshold current required to stimulate a nerve with a long pulse width.

CHRONAXIE- is the duration of the stimulus required to stimulate at twice the rheobase. Chronaxie is used to express the relative excitabilities of different tissues. It is possible to stimulate A- α (motor) fibres without stimulating A- δ and C fibres that transmit pain. Moreover, mixed nerves can be located by evoking a motor response without causing patient discomfort. Stimulation intensity will be variable as determined by coulomb's law. A very high stimulus current is required to stimulate the nerve when the needle tip is far away from the nerve. If the distance is great, the strength of the stimulus required to stimulate the



PERIPHERAL NERVE LOCATOR

nerve may produce significant pain and systemic effects. An EMR at a stimulating current of $<0.5\text{mA}$ is associated with high rates of success of PNS assisted PNB.

Characteristics of an ideal PNS:

1. Constant current output-A particular current not the voltage stimulates the nerve. Therefore, the current delivered by the device should not vary with changes in the resistance of the external circuits.
2. Digital display of the delivered current
3. Variable output control
4. Clearly identifiable polarity
5. Option for different pulses
6. A wide range of current output $0.1\text{-}5.0\text{mA}$
7. Battery indicator

Peripheral nerve stimulator settings:

- MIXED NERVE(most PNB)
 - a. Current(dial) - 1mA
 - b. Current duration - 0.1ms
 - c. Frequency - $1\text{-}2\text{Hz}$

- SENSORY NERVE

(eg- Lateral femoral cutaneous and saphenous nerves)

- a. Current (dial) - 2-5mA
- b. Current duration - 1ms
- c. Frequency - 1Hz

- DIABETIC NEUROPATHY(PNB)

- a. Current(dial) - 2mA
- b. Current duration - 0.3ms
- c. Frequency - 1-2HZ

Pharmacology

PHARMACOLOGY ^{20,43,57-59}

LOCAL ANESTHETICS

Mechanism of action of local anesthetics:

Impulse blockade by local anesthetics may be summarized by the following chronology

- Solutions of local anesthetic are deposited near the nerve. Removal of free drug molecules away from this locus is a function of tissue binding, removal by the circulation, and local hydrolysis of aminoester anesthetics. The net result is penetration of the nerve sheath by the remaining free drug molecules.
- Local anesthetic molecules then permeate the nerve's axon membranes and reside there and in the axoplasm. The speed and extent of these processes depend on a particular drug's pKa and on the lipophilicity of its base and cation species.
- Binding of local anesthetic to sites on voltage gated Na⁺ channels prevent opening of the channels by inhibiting the conformational changes that underlie channel activation. Local anesthetics bind in the channel's pore and also occlude the path of Na⁺ ions.

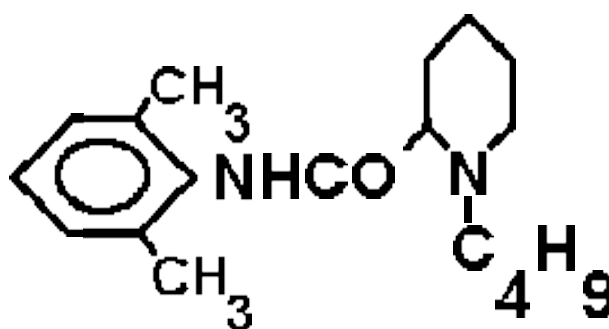
- During onset of and recovery from local anesthesia, impulse blockade is incomplete and partially blocked fibers are further inhibited by repetitive stimulation, which produces an additional, use- dependent binding to Na^+ channels
- One local anesthetic binding site on the Na^+ channel may be sufficient to account for the drugs resting and use-dependent actions. Access to this site may potentially involve multiple pathways, but for clinical local anesthetic, the primary route is the hydrophobic approach from within the axon membrane.
- The clinically observed rates of onset and recovery from blockade are governed by the relatively slow diffusion of local anesthetic molecules into and out of the whole nerve, not by their much faster binding and dissociation from ion channels. A clinically effective block that may last for hours can be accomplished with local anesthetic drugs that dissociate from Na^+ channels in a few seconds.

BUPIVACAINE HYDROCHLORIDE

It is an amide local anesthetic synthesized by B.O. Af. Evenstam in 1957 of AB Bofor in Sweden. First came into clinical use in 1963 by Widman & Teliuvo.

Chemistry:

An amino amide local anesthetic having aromatic moiety (benzene ring), which offers lipophilicity to one end of the molecule. It is linked by an amide to a tertiary amine, which is hydrophilic on the other end of the molecule. IUPAC Name: 1-butyl-N-(2,6-dimethylphenyl)piperidine-2-carboxamide



It displays stereoisomerism. Marked as a racemic mixture containing optically active enantiomers R and S. S-enantiomers have been noted to have slightly longer duration of action and lower systemic toxicity when compared to R-type.

Presentation:

As a clear solution of 0.25/05% bupivacaine hydrochloride. The hyperbaric solution contains 8-mg/ml of glucose.

Physico chemical properties:

- Molecular Weight : 288
- pKa(25 °C) : 8.1
- Protein Binding : 95%

- Lipid Solubility : 28
- Fraction nonionized at pH 7.4 - 17%
pH 7.2 - 11%

Pharmacokinetics:

Local anesthetics are weak bases with pK values somewhat above physiologic pH. As a result < 50% of the local anesthetic exists in a lipid soluble non ionized form at physiologic pH. Intrinsic vasodilator activity of bupivacaine will also influence the potency and duration of action.

The absorption of local anesthetics is related to

- The site of injection (Intercostals > Epidural> Brachial Plexus > Subcutaneous)
- The dose: a linear relationship exists between the total dose and the peak blood concentration achieved.
- The presence of vasoconstrictors which delay absorption.
- Pharmacologic characteristics of the drug.

Lipid solubility, tissue blood flow, age, cardiovascular status and hepatic function will influence the absorption and the resultant plasma concentration of the drug. Protein binding of the drug will influence the distribution and elimination. Alpha₁ acid glycoprotein is the most

important protein binding site of Bupivacaine. Volume of distribution is 73 liters.

The possible pathways for metabolism of Bupivacaine include aromatic hydroxylation, N-dealkylation, amide hydrolysis and conjugation. Only the N-dealkylated metabolite N-desbutylbupivacaine has been measured in the blood or urine. Clearance is 0.47 L/min and the elimination half time is about 210 minutes.

Routes of administration/ Doses:

Bupivacaine is used for infiltration, peripheral nerve block, epidural and spinal anesthesia.

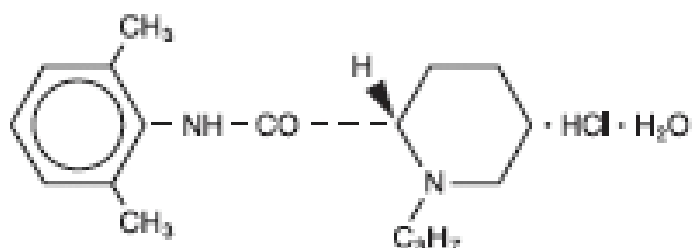
Concentration Used:

- Infiltration - 0.25%
- Peripheral nerve Block - 0.25% - 0.5%
- Epidural Anesthesia - 0.25%-0.5%
- Spinal Anesthesia - 0.5% Heavy

ROPIVACAINE HYDROCHLORIDE

Ropivacaine Hydrochloride is a member of the amino amide class of local anesthetics and is supplied as the pure S-(-)-enantiomer. Ropivacaine HCl is chemically described as S-(-)-1-propyl-

2',6'-pipecoloxylidide hydrochloride monohydrate. The drug substance is a white crystalline powder, with a molecular formula of $C_{17}H_{26}N_2O \cdot HCl \cdot H_2O$ and the following structural formula:



Physico chemical properties:

- Molecular weight - 328.89
- pKa - 8.1
- Protein binding - 94%
- Fraction nonionized % at pH 7.4 -17

Pharmacokinetics:

Ropivacaine is 94% protein bound, mainly to Alpha₁-acid glycoprotein. The lipid solubility is intermediate between lidocaine and bupivacaine. Volume of distribution is 59 liters. Ropivacaine is extensively metabolized in the liver, predominantly by aromatic hydroxylation. It is metabolized to 2,6-pipecoloxylidide and 3-hydroxyropivacaine by hepatic cytochrome P-450 enzymes. Only a small fraction of ropivacaine is excreted unchanged in the urine. Clearance is 0.44 (L/min) and elimination half time is 108 min. Overall, the clearance

of ropivacaine is higher than that determined for bupivacaine and its elimination half –time is shorter. The higher clearance of ropivacaine may offer an advantage over bupivacaine in terms of systemic toxicity.

Presentation:

As a clear solution of 0.25% /0.5%/0.75% Ropivacaine (Naropin) hydrochloride.

Routes of administration / Doses:

- Infiltration - 2.5 and 5 mg/ml
- Peripheral nerve blockade - 5 and 10 mg/ml
- Epidural - 5 and 7.5 mg/ml

SIDE EFFECTS OF LOCAL ANESTHETICS:

The principal side effects related to the use of local anesthetics are allergic reactions and systemic toxicity due to excessive plasma concentrations of the local anesthetic.

Allergic reactions:

Allergic reactions to the amide type local anesthetics are extremely rare when compared with esters of local anesthetics due to the metabolite paraaminobenzoic acid. Allergic reaction may also be due to

methylparaben or similar substances used as preservatives in commercial preparations of ester and amide local anesthetics.

Systemic toxicity:

Systemic toxicity of local anesthetics is due to an excess plasma concentration of the drug, accidental intravascular injection. Systemic toxicity of local anesthetics involves the central nervous system and cardiovascular system.

Central nervous System:

The principal effect of Bupivacaine is reversible neural blockade. This leads to a characteristically biphasic effect in the central nervous system. During accidental over dosage or direct vascular injections the clinical signs are numbness of tongue and circumoral tissue, light headedness, visual and auditory disturbances, muscular twitching and tremors. Skeletal muscle twitching is often first evident in the face and extremities and signal immediate tonic- clonic seizures. The signs may progress to generalized convulsion of the tonic clonic nature. When plasma levels continue to rise, CNS excitation is rapidly superseded by depression (drowsiness, disorientation and coma). The typical plasma concentration of Bupivacaine associated with seizure is 4.5 to 5.5 ug/ml. In studies which subjected volunteers to continuous intravenous infusions of ropivacaine and bupivacaine until the onset of CNS symptoms,

volunteers tolerated a 25% greater total dose of ropivacaine than bupivacaine, and plasma levels of ropivacaine were greater at onset of symptoms.⁹⁰

Cardiovascular System

The cardiovascular system is more resistant to the toxic effects of high plasma concentrations of local anesthetics than is the CNS. Bupivacaine is markedly cardiotoxic. It results from blockade of cardiac sodium channels. At high plasma concentrations sufficient cardiac sodium channel is blocked so that conduction and automaticity becomes adversely depressed. Effects of the drug on calcium ion and potassium ion channels and local anesthetic induced inhibition of cyclic adenosine monophosphate (cAMP) production may also contribute to cardio toxicity. Accidental IV injection may result in precipitous hypotension, cardiac dysrhythmias and atrioventricular heart block. It may cause ventricular cardiac dysrhythmias through direct brain stem effect. Cardiotoxic plasma concentration of Bupivacaine is 8-10 ug/ml.

Commercial bupivacaine is a 50:50 racemic mixture of the S- and R-enantiomers. Because of its greater affinity for and dwell time at voltage-gated sodium channels, the R configuration confers greater cardiotoxicity to racemic bupivacaine. Compared to the S-enantiomer, R-bupivacaine binds three times more firmly to the sodium channel, and

unbinds 4.4 times as slowly. R-bupivacaine is also more arrhythmogenic, and slows ventricular conduction 4.6 times as much as S-bupivacaine. Ropivacaine is manufactured as the pure S-enantiomer in order to take advantage of the decreased cardiotoxicity of the S-configuration. The decrease in cardiotoxicity is due to S-ropivacaine's stereoselectivity.

USE OF VASOCONSTRICTORS

Epinephrine (1:2,00,000 or 5µg/ml) when added to local anesthetic solution, produce vasoconstriction which limits systemic absorption and maintains the drug concentration in the vicinity of the nerve fibres to be anesthetized. Thus it prolongs the duration of block and also by lowering the peak blood level, it reduces the incidence of systemic toxicity of the local anaesthetic. Most local anesthetics with the exception of ropivacaine possess intrinsic vasodilator properties and it is possible that epinephrine induced vasoconstriction will slow clearance from injection site and thus prolonging the time the drug is in contact with the nerve fibres.

The extent to which epinephrine prolongs the duration of anesthesia depends on the specific local anesthetic used and the site of injection. In addition to decreasing systemic absorption to prolong conduction blockade, it may also enhance conduction blockade by increasing neuronal uptake of local anesthetic. The alpha-adrenergic effects of epinephrine may be associated with some degree of analgesia

that could contribute to the effects of conduction blockade. The addition of epinephrine to local anesthetic solution has little, if any, effect on the onset rate of local analgesia.

Systemic absorption of epinephrine may accentuate systemic hypertension in vulnerable patients.

EPINEPHRINE

It's a non selective agonistic of all adrenergic receptor including α_1 , α_2 , β_1 , β_2 and β_3 receptors. Epinephrine is synthesized in the adrenal gland in an enzymatic pathway that converts amino acid tyrosine in a series of intermediates and ultimately epinephrine. Tyrosine is first oxidized to L-dopa, which is subsequently decarboxylated to give dopamine. Dopamine on oxidation gives norepinephrine, which is methylated to give epinephrine.

Formula: $C_9H_{13}NO_3$

Functions:

- Regulation of myocardial contractility, heart rate, vascular and bronchial smooth muscle tone.
- Potentiates glandular secretions and metabolic processes like glycogenolysis and lipolysis.

Pharmacokinetics:

Oral administration is not effective because epinephrine is rapidly metabolized in the gastrointestinal mucosa and liver. Absorption after SC injection is slow because of local epinephrine induced vasoconstriction. Epinephrine is poorly lipid soluble, preventing its ready entrance into the CNS and accounting for the lack of cerebral effects. Half life is 2 minutes.

Clinical uses:

- Addition to local anesthetic solutions to decrease systemic absorption and prolong the duration of action of the anesthetic
- Treatment of life-threatening allergic reaction
- During cardiopulmonary resuscitation as the single most important therapeutic drug, and
- Continuous infusion to increase myocardial contractility.

Side effects:

Palpitation, Tachycardia, Anxiety, Headache, Tremors, Hypertension, Acute pulmonary oedema.

Review of Literature

REVIEW OF LITERATURE

1. A comparison of ropivacaine 0.5% and bupivacaine 0.5% for brachial plexus block. Anesthesiology. 1991 Apr; 74(4):639-42. Hickey R, Hoffman J, Ramamurthy S.

Forty-eight patients received a subclavian perivascular brachial plexus block for upper-extremity surgery. One group (n = 24) received ropivacaine 0.5% (175 mg) and a second group (n = 24) received bupivacaine 0.5% (175 mg), both without epinephrine. Onset times for analgesia and anesthesia in each of the C5 through T1 brachial plexus dermatomes did not differ significantly between groups. Duration of analgesia and anesthesia was long (mean duration of analgesia, 13-14 h; mean duration of anesthesia, 9-11 h) and also did not differ significantly between groups. Motor block was profound, with shoulder paralysis as well as hand paresis developing in all of the patients in both groups. Two patients in each group required supplemental blocks before surgery.

Conclusion: Ropivacaine 0.5% and bupivacaine 0.5% appeared equally effective in providing brachial plexus anesthesia.

2. 0.75% and 0.5% ropivacaine for axillary brachial plexus block: A clinical comparison with 0.5% bupivacaine. Reg Anesth Pain Med 1999; 24:514-8. Laura Bertini M.D, Vincenzo Tagariello M.D., Stefania Mancini M.D., et al

It's a double blinded , randomized , prospective study . 32 mL of the local anesthetic solution into the midaxilla, by a nerve-stimulator technique. The rate of complete sensory and motor block observed with both ropivacaine groups was higher at 10, 15, and 20 minutes post injection ($P < .001$). The mean peak time was shorter with ropivacaine than with bupivacaine ($R_{50} = 16.37$ minutes, $R_{75} = 14.7$ minutes, $B = 22.3$ minutes, $P < .05$). The quality of the anesthesia was higher with ropivacaine, as measured by the intraoperative needs for opioids and the overall patient's satisfaction ($P < .05$). **Conclusions:** Ropivacaine showed advantages over bupivacaine for axillary brachial plexus block. Because no statistical differences were found between the two ropivacaine groups, we therefore conclude that 0.75% does not add benefit and that 0.5% ropivacaine should be used to perform axillary brachial plexus blocks.

3. Brachial plexus block with a new local anaesthetic: 0.5 per cent ropivacaine. Can J Anaesth. 1990 Oct; 37(7):732-8. Hickey R, Candido KD, Ramamurthy S, Winnie AP et al

0.5 % ropivacaine was used in 32 patients receiving a subclavian perivascular block for upper extremity surgery. One group ($n = 15$) received 0.5 per cent ropivacaine without epinephrine and a second group ($n = 17$) received 0.5 per cent ropivacaine with epinephrine in a concentration of 1:200,000. Anaesthesia was achieved in 87 per cent of the patients in both groups in all of the C5 through T1 brachial plexus

dermatomes. Motor block was profound with 100 per cent of patients in both groups developing paresis at both the shoulder and hand and 100 per cent developing paralysis at the shoulder. There was a rapid initial onset of sensory block (a mean of less than four minutes for analgesia) with a prolonged duration (a mean of greater than 13 hr of analgesia). The addition of epinephrine did not significantly affect the quality or onset of sensory or motor block. The duration of sensory block was reduced by epinephrine at T1 for analgesia and at C7, C8, and T1 for anaesthesia. The duration of sensory block in the remaining brachial plexus dermatomes as well as the duration of motor block was not affected by epinephrine. There was no evidence of cardiovascular or central nervous system toxicity in either group with a mean dose of 2.5-2.6 mg/kg ropivacaine.

4. Comparison of S (-) Bupivacaine and racemic (RS) - bupivacaine in supraclavicular brachial plexus block. BJA 1998; 80:594- 598. C.R Cox, M.R. Checketts, N. Mackenzie, N.B.Scott et al

This study compared S (-) Bupivacaine with racemic RS - bupivacaine in supraclavicular brachial plexus block in 75 patients undergoing elective hand surgery. Patients received 0.4 ml/kg of either 0.25% or 0.5% S (-) bupivacaine or 0.5% RS bupivacaine in a randomized double blind study. There were no significant differences in onset time, dermatomal spread or duration of sensory and motor block

between three groups. Duration of sensory block was prolonged with wide inter- patient variation. **Conclusion:** S (-) bupivacaine was suitable for local anesthetic use in brachial plexus block.

5. A comparative study of 0.25% ropivacaine and 0.25% bupivacaine for brachial plexus block. Anesth-Analg. 1992 Oct. 75(4) 602-6. Hickey.R,Rowley.C.L, Ramamurthy.S, Winnie.A.P et al

The randomized double blind study compares the effectiveness of 0.25% ropivacaine and 0.25% bupivacaine in 44 patients receiving a subclavian perivascular brachial plexus block for upper extremity surgery.. Onset times for analgesia and anesthesia did not differ significantly between the two groups. The mean onset time for analgesia ranged from 11.2 to 20.2 min, and the mean onset time for anesthesia ranged from 23.3 to 48.2 min. The onset of motor block differed only with respect to paresis in the hand, with bupivacaine demonstrating a shorter onset time than ropivacaine. The duration of sensory and motor block also was not significantly different between the two groups. The mean duration of analgesia ranged from 9.2 to 13.0 h, and the mean duration of anesthesia ranged from 5.0 to 10.2 h. Both groups required supplementation with peripheral nerve blocks or general anesthesia in a large number of cases. **Conclusion:** In view of the frequent need for supplementation noted with both 0.25% ropivacaine and 0.25%

bupivacaine, we do not recommend using the 0.25% concentrations of these local anesthetics to provide brachial plexus block.

6. A Comparison of 0.5% Bupivacaine, 0.5% Ropivacaine, and 0.75% Ropivacaine for Interscalene Brachial Plexus Block. *Anesth Analg* 1998; 87:1316-9. Stephen M. Klein, Roy A. Greengrass, Susan M. Steele, Fran J. D'Ercole, Kevin P. Speer, David H. Gleason, et al

Seventy-five adult patients were entered into this double-blind, randomized study. Patients were assigned (n = 25 per group) to receive an interscalene block using 30 ml of 0.5% bupivacaine, 0.5% ropivacaine, or 0.75% ropivacaine. All solutions contained fresh epinephrine in a 1:400,000 concentration. The mean onset time of both motor and sensory blockade was <6 min in all groups. Duration of sensory blockade was similar in all groups as defined by the three recovery measures.

Conclusion: There is no clinically important difference in times to onset and recovery of interscalene block for Bupivacaine 0.5%, ropivacaine 0.5%, and ropivacaine 0.75% when injected in equal volumes. In addition, increasing the concentration of ropivacaine from 0.5% to 0.75% fails to improve the onset or duration of interscalene brachial plexus block.

7. A clinical comparison of ropivacaine 0.75%, ropivacaine 1% or bupivacaine 0.5% for interscalene brachial plexus anaesthesia.

**European Journal of Anaesthesiology (1999), 16: 11: 784-789. A
Casati, G Fanelli, G. Cappelleri, P. Beccari, L. Magistris, et al**

45 patients, undergoing elective shoulder surgery, were randomly allocated to receive interscalene brachial plexus anaesthesia with 20 mL of either ropivacaine 0.75% (n=15), ropivacaine 1% (n=15), or bupivacaine 0.5% (n=15). Readiness for surgery was achieved later with bupivacaine 0.5% (28 ± 15 min) than with ropivacaine 1% (10 ± 5 min) ($P=0.005$) and ropivacaine 0.75% (15 ± 8 min) ($P=0.0005$). 7 patients receiving bupivacaine 0.5% required intra-operative analgesic supplementation (fentanyl 0.1 mg intravenous) compared with one patient receiving ropivacaine 0.75%, and two patients treated with ropivacaine 1% ($P=0.02$). The time from the block placement to first request for pain medication was similar in the three groups. **Conclusion:** 0.75% or 1% ropivacaine allows for a prolonged post-operative pain relief, similar to that provided by bupivacaine 0.5%, with short onset time of surgical anaesthesia.

**8. A Clinical and Pharmacokinetic Comparison of Ropivacaine and Bupivacaine in Axillary Plexus Block. Anesth Analg 1995; 81:534-8.
Vilho A. Vainionpaa G et al**

The clinical and pharmacokinetic properties of ropivacaine and bupivacaine, both 5 mg/mL, used in axillary plexus block were compared

in 60 patients in this randomized, double-blind, parallel-group study. The axillary plexus was identified with a nerve stimulator and 30, 35, or 40 mL of drug, depending on body weight, was injected into the perivascular sheath. In 20 patients, venous blood samples for the pharmacokinetic measurement were obtained over 24 h. The median onset time for anesthesia and complete motor block were in the range of 12-48 min and 5-20 min, respectively. Thirty-eight percent of patients in the ropivacaine group and 29% in the bupivacaine group needed additional nerve block(s) or supplementary analgesia and 7% in the bupivacaine group needed general anesthesia for surgery. Anesthesia was achieved in 52%-86% of the evaluated six nerves in the ropivacaine group and in 36%-87% in the bupivacaine group; the lowest figures were seen in the musculocutaneous nerve. In the pharmacokinetic study the mean peak plasma concentrations were 1.28 ± 0.21 mg/L in the ropivacaine group and 1.28 ± 0.47 mg/L in the bupivacaine group and the median times to peak plasma concentration were 0.86 h and 0.96 h, respectively. **Conclusion:** No statistically significant differences were found between ropivacaine and bupivacaine in either the clinical or the pharmacokinetic comparisons.

9. Plasma concentrations of ropivacaine given with or without epinephrine for brachial plexus block. Can J Anaesth 1990 / 37:8 / pp878-82. Rosemary Hickey, Janna Blanchard, Joan Hoffman, Jan Sjøvall, Somayaji Ramamurthy.

Single injection of 33 ml ropivacaine for subclavian perivascular block and 5 ml to block the intercostobrachial nerve in the axilla was given. One group (n = 8) received 0.5 per cent ropivacaine without epinephrine and the other (n = 9) received 0.5 per cent ropivacaine with epinephrine 1:200,000. Plasma ropivacaine concentrations were measured from peripheral venous blood samples taken for 12 hr after drug administration. The mean peak plasma concentration was 1.6 ± 0.6 mg/L and 1.3 ± 0.4 mg /L after administration of ropivacaine with and without epinephrine. The median time to peak plasma concentration was 0.75 hr and 0.88 hr. The differences were not statistically significant.

Conclusion: Addition of epinephrine does not alter tire pharmacokinetic properties of ropivacaine when used for subclavian perivascular brachial plexus block.

10. Epinephrine Does Not Prolong the Analgesia of 20 mL Ropivacaine 0.5% or 0.2% in a Femoral Three-In-One Block. Anesth Analg. 1988 Nov; 67(11):1053-8. Anne Weber, Roxane Fournier, Elisabeth Van Gessel, Nicolas Riand et al

41 patients undergoing total knee replacement under combined peripheral block/general anesthesia were randomly allocated to two groups. After insertion of a femoral catheter, 21 patients in the Ropivacaine-Epinephrine (ROPI-EPI) group received 20 mL ropivacaine 0.5% plus epinephrine 1:200,000, whereas 20 patients in the Ropivacaine

group (ROPI) received 20 mL plain ropivacaine 0.5%. Thereafter, a sciatic block with 30 mL bupivacaine 0.5% plus epinephrine 1:200,000 was performed in all patients, followed by general anesthesia. After surgery, patient-controlled analgesia (PCA) with ropivacaine 0.2% plus epinephrine 1:200,000 for Group ROPI-EPI and plain ropivacaine 0.2% for Group ROPI was available via the femoral catheter (200 mL ropivacaine 0.2% \pm epinephrine, bolus 20 mL, lockout 120 min). The interval between the initial ropivacaine injection and the first PCA injection determined the duration of 20 mL ropivacaine 0.5% \pm epinephrine, whereas the interval between the first and second PCA injection determined the duration of 20 mL ropivacaine 0.2% \pm epinephrine. The average duration of ropivacaine 0.5% was 657 ± 345 min for the ROPI-EPI group and 718 ± 423 min for the ROPI group (NS), whereas for ropivacaine 0.2%, the average duration was 409 ± 245 min for the ROPI-EPI group and 419 ± 339 min for the ROPI group (not significant). **Conclusion:** We conclude that epinephrine does not influence the duration of analgesia of the ropivacaine concentrations investigated.

11. High-dose bupivacaine, levobupivacaine and ropivacaine in axillary brachial plexus block. Acta Anaesthesiologica Scandinavica [2004, 48(5):601-6]. Liisanantti O, Luukkonen J, Rosenberg PH

In 90 patients scheduled for hand and forearm surgery, a perivascular axillary brachial plexus block was performed with 45 ml of 5 mg/ml of either racemic bupivacaine-HCl, levobupivacaine-HCl, or ropivacaine-HCl. After similar onsets of sensory block, the sum of completely anaesthetized innervation areas of the four main nerves at 45 min was greater in the ropivacaine group than in the levobupivacaine group ($P < 0.01$). Complete motor block at the elbow was more frequent in the ropivacaine group (67%) than in the bupivacaine (47%) and levobupivacaine groups (30%). In the hand, the corresponding results were 83%, 77%, and 57%, respectively. Mean duration of the blocks was similar in the bupivacaine, levobupivacaine and ropivacaine groups at 19.3 h, 19.5 h, and 17.3h, respectively. **Conclusion:** Ropivacaine-HCl 5 mg/ml produced slightly better sensory and motor block intensity than the same dose of levobupivacaine-HCl.

12. Ropivacaine: An Update of its Use in Regional Anaesthesia.

Drugs: November 2000 - Volume 60 - Issue 5 - pp 1065-1093.

McClellan, Karen J.; Faulds, Diana

In patients about to undergo upper limb surgery, 30 to 40ml ropivacaine 0.5% produced brachial plexus anaesthesia broadly similar to that achieved with equivalent volumes of bupivacaine 0.5%, although the time to onset of sensory block tended to be faster and the duration of motor block shorter with ropivacaine. **Conclusion:** Ropivacaine is a well

tolerated regional anaesthetic with an efficacy broadly similar to that of bupivacaine. However, it may be a preferred option because of its reduced CNS and cardiotoxic potential and its lower propensity for motor block

13. The place of ropivacaine in anesthesia. Acta Anaesth. Belg., 2003, 54, 141-148. R. STIENSTRA.

Ropivacaine has a lower systemic toxicity than both racemic and levobupivacaine. Especially its better cardiotoxic profile has been well documented and is an important advantage when using techniques with a potential for high plasma concentrations. The lower systemic toxicity of ropivacaine compared to bupivacaine is not offset by a lower potency, as ropivacaine in a 50% higher dose is still less cardiotoxic.

14. The subclavian perivascular technique of brachial plexus anesthesia. Aneesthesiology 1964; 25:353-363. Winnie A, Collins V

The prevertebral fascia envelops the brachial plexus from the cervical vertebrae to the distal axilla, forming a subclavian perivascular space that is continuous with the axillary perivascular space. By applying the concept of the axillary perivascular technique to the supraclavicular approach, subclavian perivascular technique was developed which affords greater simplicity, safety and consistency of results. Once the space is

entered a single injection is necessary and the extent of anesthesia will depend on the volume of anesthetic and the level at which it is injected.

15. The Supraclavicular Block with a Nerve Stimulator: To Decrease or Not to Decrease, That Is the Question. *Anesth Analg* 2004; 98:1167–71. Carlo D. Franco, Vitaliy Domashevich, Gennadiy Voronov, Amir B. Rafizad, and Tanyu J. Jelev

This study concluded that during the performance of a supraclavicular block eliciting a clearly visible response of the fingers at 0.9 mA can be immediately followed by the injection of local anesthetic, because decreasing the output to 0.5 mA does not seem to improve the overall quality of the block as measured by the onset and duration of anesthesia or patient satisfaction.

Material and Methods

MATERIALS AND METHODS

This is a prospective randomized double blinded study conducted at Government Stanley Hospital, attached to Stanley Medical College, Chennai from April 2010 to October 2010 after approval by the departmental dissertation committee and the hospital's ethical committee. Seventy five patients of ASA grade I or II of either sex undergoing plastic surgery (both elective and emergency) on forearm or hand were randomly allocated into three groups. Each group comprises of 25 patients.

Inclusion criteria:

- Age 18 - 45 yrs
- Sex (Male/ Female)
- Weight (50-65 kg)
- ASA grade I & II scheduled to undergo forearm and hand surgery under subclavian perivascular brachial plexus block

Exclusion criteria

- Coagulopathy
- Infection at the puncture site
- Hypertension
- Diabetes mellitus
- Allergy to amide local anesthetics

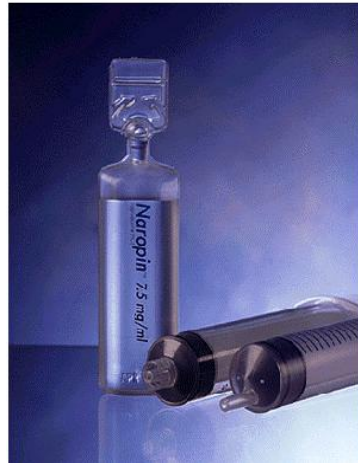
- Pregnancy
- Severe pulmonary pathology
- Mental incapacity or language barrier
- BMI more than 35
- Anatomical variations

Drugs and Equipment:

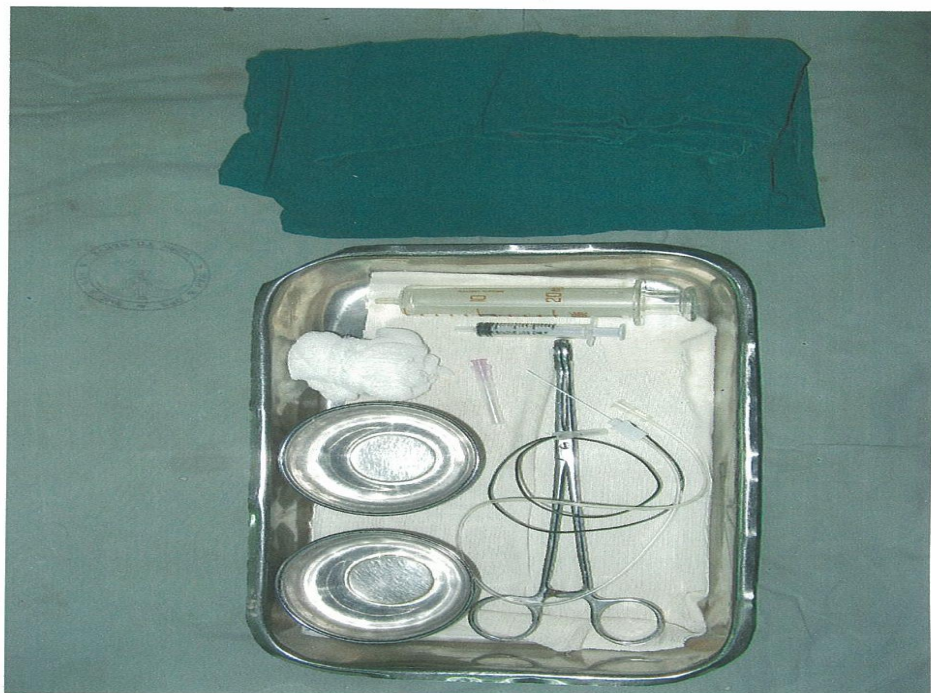
- Oxygen source
- BRAUN –Stimuplex DIG – Nerve locator
- Disposable Braun – Stimuplex (insulated) needle A50(22Gx2’')
- 0.5 % Bupivacaine and 0.5% Ropivacaine
- Epinephrine (1:2,00,000)
- Midazolam and Fentanyl injection
- Appropriate size intravenous cannula and IV fluids
- Standard monitors
- Airway equipments
- All emergency drugs.

Procedure

All patients were preoperatively evaluated for any systemic diseases and investigations done prior to assessment. Procedure was



DRUGS



EQUIPMENTS

Equipments

explained in detail and written consent was obtained. Procedure was carried out in theatre where facilities for resuscitation were available.

After obtaining informed consent, patients were randomly assigned to receive 30 ml of one of three different solutions.

- GROUP I – Receives 0.5 % Ropivacaine without epinephrine.
- GROUP II-Receives 0.5% Ropivacaine with epinephrine (1:2,00,000)
- GROUP III – Receives 0.5% bupivacaine.

0.5% ropivacaine was prepared by adding 10 ml of distilled water to 20 cc of 0.75% ropivacaine (ROPIN 0.75% - 7.5 mg/l, Preservative free 20 ml amp). Epinephrine is available as 1 mg in 1ml amp. 9 ml of distilled water is added to it thus each ml now contains 100 micro gram. 1.5 ml of this is added along with 8.5 ml of distilled water to available 0.75% ropivacaine to get 30 ml of 0.5% Ropivacaine with epinephrine(1:2,00,000). The randomization involved the use of sealed envelopes. A sealed envelope was randomly selected by the patient and opened by an assistant, with instructions to draw up the relevant drug. The syringe was labelled with the patient's name and it was handed to the investigator.

All patients were brought to preoperative holding area. After connecting monitors to the patient, Intravenous line was started for all the

patients in the contralateral arm with 18G intravenous cannula and IV fluid was started. Patient was sedated with IV Midazolam 0.01 to 0.02 mg/kg and Fentanyl 1 to 2 microgram / kg. Each patient received subclavian perivascular brachial plexus block using nerve locator.

SUBCLAVIAN PERIVASCULAR BRACHIAL PLEXUS BLOCK:

Patient was placed on the table and proper illumination was done at the site of block.

POSITION:

- Patient was placed in supine position with head turned 30⁰ to the opposite side to be injected.
- The arms were placed at the patient's side with hands pointing towards the knee. The arm on the side to be injected may be pulled to depress the clavicle and the shoulder.
- A rolled towel was placed lengthwise between the shoulders along the spine to give the best exposure of the area.

STEPS:

The block site was aseptically prepared and draped.

- The anesthesiologist stands at the head end of the table.



NEEDLE INSERTION FOR SUBCLAVIAN PERIVASCULAR BLOCK



FINGER AND THUMB FLEXION - THE IDEAL RESPONSE TO NERVE STIMULATION

- The patient was asked to lift the head slightly to bring the clavicular head of the sternomastoid muscle into prominence.
- The index finger was placed lateral to the muscle and the patient is asked to relax. The index finger was rolled laterally across the belly of the muscle until the interscalene groove is palpated.
- Finger was then moved inferiorly down the groove until the pulse of the subclavian artery was palpated between the scalene muscles.
- A skin wheal was raised at this point with 2 ml of 2 % lignocaine with a 24 G needle about 2-3 cms above the midpoint of the clavicle.
- Pulsation of the subclavian artery against the palpating finger is a guide to supraclavicular block.

Positive pole of the cable is connected to the patient's arm on the side of block. Negative pole of the cable is connected to stimulating block needle. Injecting syringe with local anesthetic solution is connected with extension catheter of the block needle. Then, with the nerve stimulator output set at 0.9 mA at 1 Hz, the needle was inserted just above the palpating finger and was advanced directly caudad (parallel to the table) until a desired evoked motor response of flexion or extension of all the

fingers was obtained, at which point the output was reduced to 0.5 mA. If the response was still visible at this level of stimulation, the needle is stabilized and 2-3ml of local anesthetic is injected after a negative aspiration for blood according to the group

- Group I patient receives 30 ml of 0.5% of ropivacaine without epinephrine,
- Group II patient receives 30 ml of 0.5% of ropivacaine with epinephrine (1:2,00,000)
- Group III patient receives 30 ml of 0.5% bupivacaine.

If the needle is in the perivascular space, the volume of the local anesthetic will produce a “pressure paraesthesia”. The rest of the local anesthetic should then be injected with frequent aspiration to prevent an intravascular injection.

- If the needle penetrates the artery it simply indicates that it is too far anterior.
- The intercostobrachial nerve was blocked separately at the axilla anterior to the axillary artery by subcutaneous injection of local anesthetic to ensure complete anesthesia of the upper extremity

- The needle should not be advanced beyond 2.5 cm to avoid the risk of complications (cervical cord injury, pneumothorax, carotid artery puncture) A cough by the patient is a warning that the pleura is being irritated by the needle.

After injecting the local anesthetic, the block was tested for both sensory (using pin prick) and motor (using muscle power). Motor block was evaluated by thumb abduction (Radial nerve), thumb adduction (Ulnar nerve), thumb opposition (Median nerve) and flexion of the elbow in supination and pronation of the forearm (Musculocutaneous nerve).

The following grade was used in the study for assessing sensory and motor blockade.

Sensory blockade

- Grade 0 = No loss of sensation to pin prick
- Grade 1 = Analgesia (Patient feels touch, but not pain)
- Grade 2 = Anesthesia (Patient does not feel touch)

Motor blockade

- Grade 0 = No weakness
- Grade 1 = Paresis (loss of wrist or elbow flexion)
- Grade 2 = Paralysis (inability to move the limb)

Evaluation of sensory and motor function was carried out prior to block and then every minute till the onset of blockade and thereafter every 5 min till one hour and then hourly until complete recovery.

PARAMETERS OBSERVED:

- Onset of sensory blockade: Onset of sensory blockade is considered when grade 1 is achieved.
- Onset of motor blockade: Onset of motor blockade is considered when grade 1 is achieved.
- Duration of sensory blockade: Duration of sensory blockade is defined as a time interval between the onset of the sensory blockade till attainment of grade 0.
- Duration of motor blockade: Duration of the motor block is defined as the time interval between the onset of motor block till attainment of grade 0.
- Vital signs: In addition patients is monitored continuously for any signs of cardiovascular or CNS toxicity. Heart rate, systolic, diastolic and mean arterial pressure is monitored every ten minutes for one hour, every 30 min till three hours, every three hours till 12 hours then sixth hourly till 24 hours.

- Complications (Pneumothorax, accidental vascular puncture, hematoma) and side effects (nausea, vomiting, convulsions, shivering, pruritus, perioral numbness) are looked for.

Statistical analysis:

Data's like age weight and onset and duration of sensory and motor block are analyzed using analysis of variance (ANOVA)

Sex is analyzed using Chi square test.

P value <0.05 is taken as statistically significant.

Mean values of hemodynamic parameters (heart rate, systolic BP, diastolic BP and Mean arterial BP) are analyzed over a period of six hours after giving block. These are compared with the base line values. Any deviation of more than 30 % from the base line values is considered as hemodynamic stability^{82,83}.

Observation & Results

OBSERVATION AND RESULTS

In this prospective randomized double blind study we compared the safety and efficacy of 0.5% Ropivacaine without epinephrine (**group I**) and 0.5% Ropivacaine with epinephrine (**group II**) and 0.5% Bupivacaine (**group III**) in patients receiving subclavian perivascular brachial plexus block in seventy five patients(twenty five in each group).

TABLE 1: AGEWISE COMPARISON

Age	Group I	Group II	Group III
Mean	26.60	27.52	25.32
Range	18-45	18-42	18-45
SD	8.52	7.33	7.57
F value	0.50		
p value	0.61(not significant)		

The mean age group of patient is 26.60 ± 8.52 SD, 27.52 ± 7.33 SD and 25.32 ± 7.57 SD in group I, group II and group III respectively. The difference is not significant statistically ($p > 0.05$).

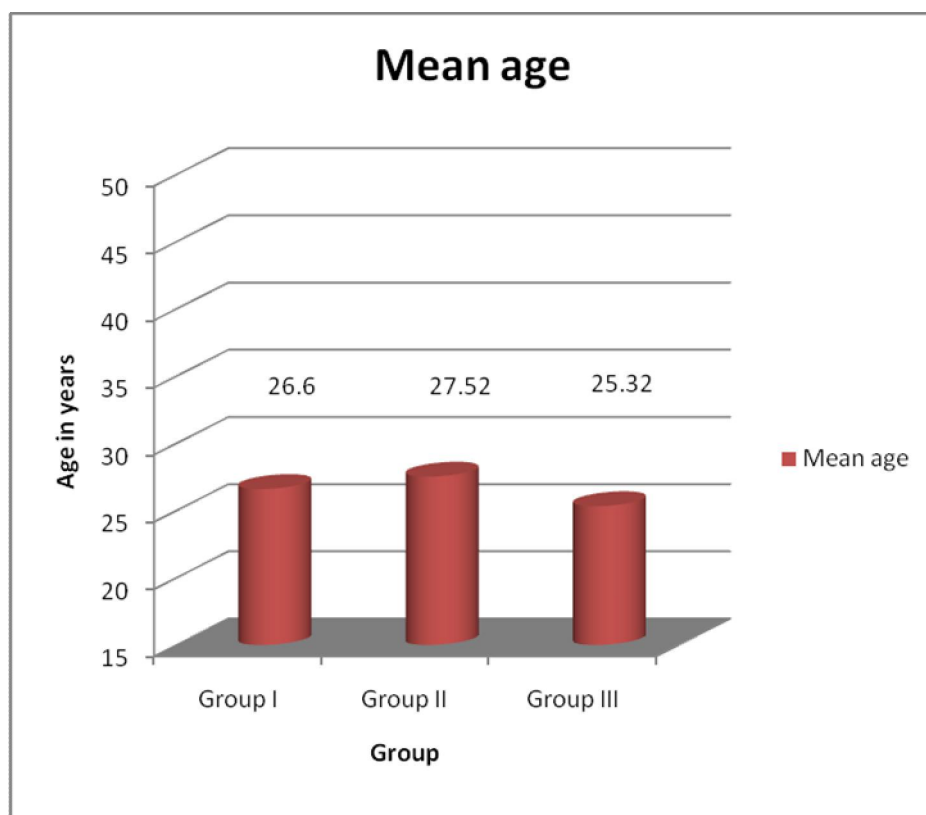


TABLE 2: SEX DISTRIBUTION

Sex	Group I		Group II		Group III		Total	
	No	%	No	%	No	%	No	%
Male	19	76	14	56	21	84	54	72
Female	06	24	11	44	04	16	21	28
Total	25	100	25	100	25	100	75	100

There are 19 male and 6 females in group I; 14 Males and 11 females in group II and 21 males and 4 females in group III. p value is 0.08 The sex distribution between groups is not statistically significant ($p > 0.05$)

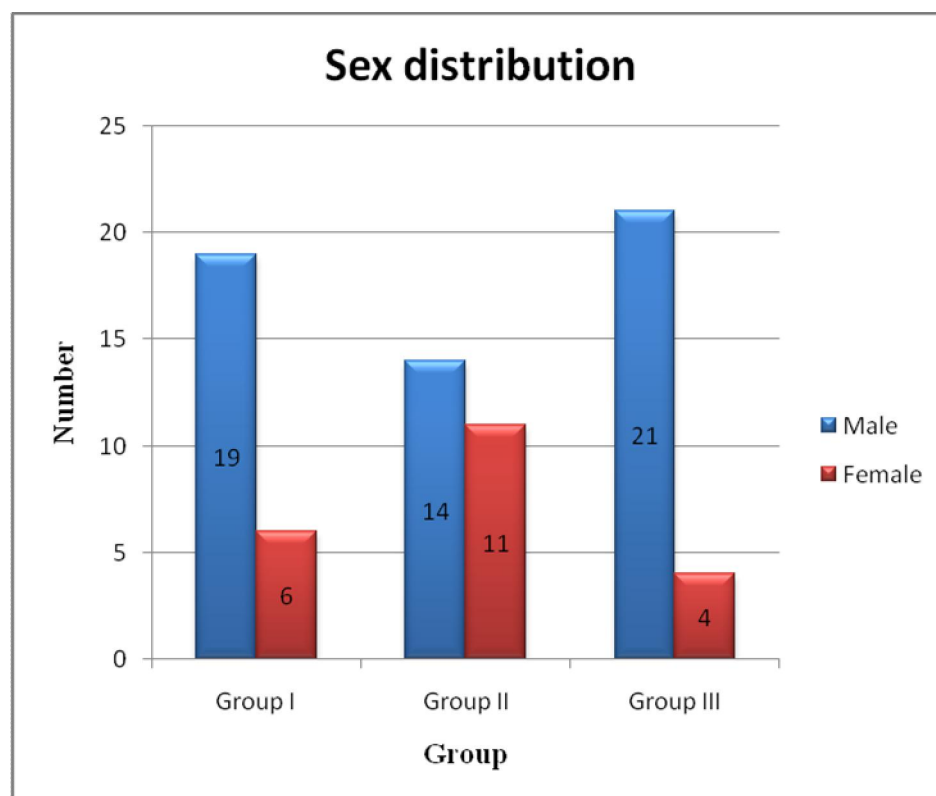


TABLE3: WEIGHT

Weight (kg)	Group I	Group II	Group III
Mean	60.20	60.04	61.40
Range	50-65	50-65	51-65
SD	5.06	5.25	3.70
p value	0.54 (not significant)		

The mean weight of patient is 60.20 ± 5.06 SD, 60.04 ± 5.25 SD and 61.40 ± 3.70 SD in group I, group II and group III respectively. The difference is not significant statistically ($p > 0.05$).

Mean weight

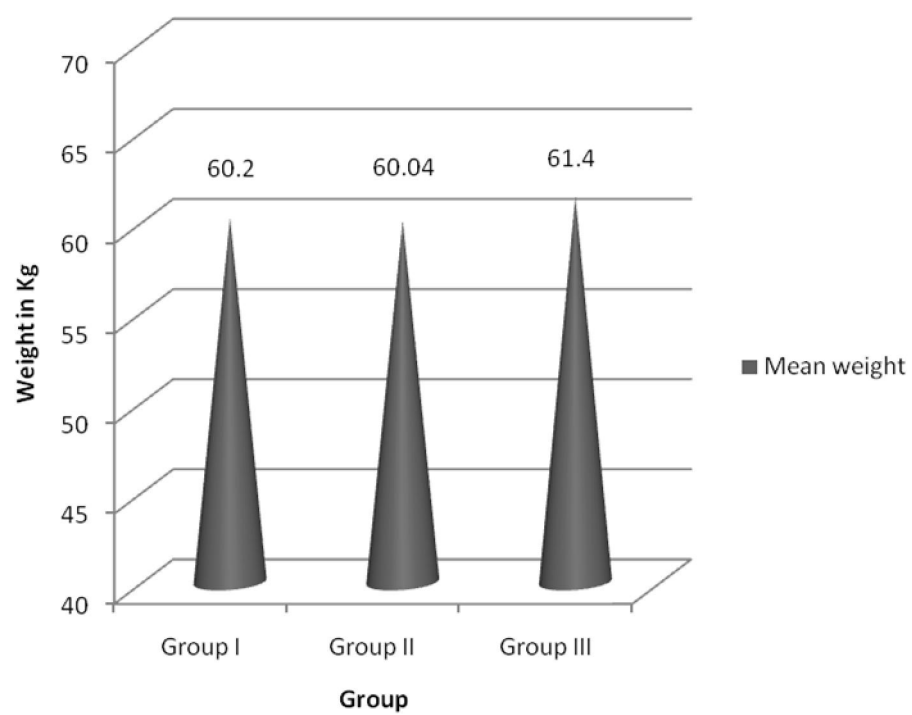


TABLE 4. ONSET OF SENSORY BLOCK

Onset of sensory block (min)	Group I	Group II	Group III
Mean	8.32	8.36	8.40
Range	7-15	5-12	7-10
SD	1.57	1.29	0.82
F value	0.03		
p value	0.98 (not significant)		

The onset of sensory block is 8.32 min, 8.36 min and 8.40 min respectively for group I, group II and group III respectively. This is not statistically significant between groups ($p > 0.05$).

Onset of sensory block

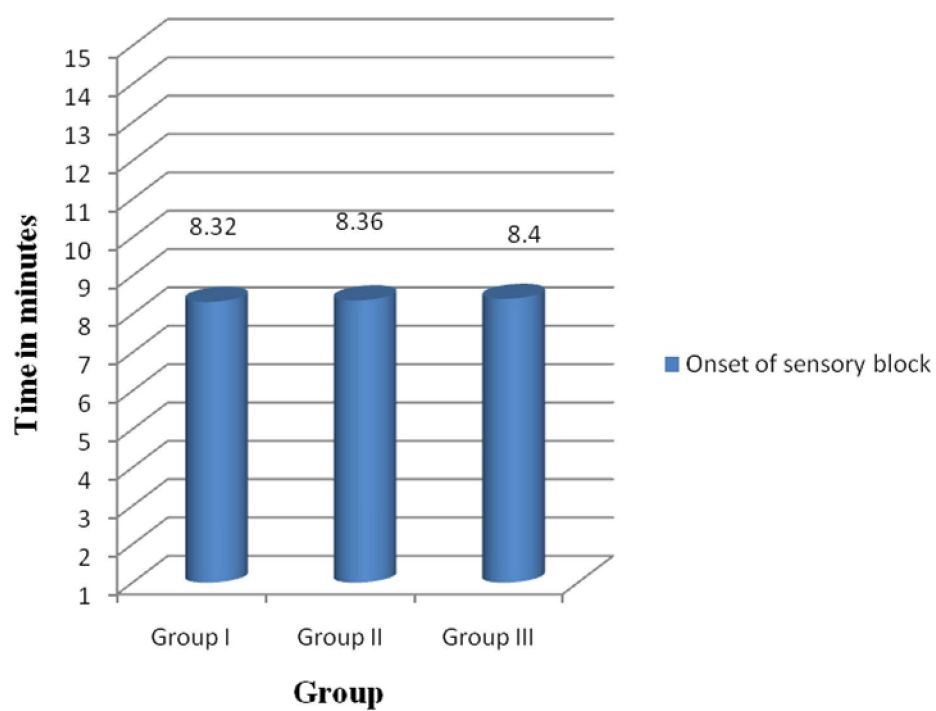


TABLE 5. ONSET OF MOTOR BLOCK

Onset of motor block (min)	Group I	Group II	Group III
Mean	6.84	7.24	6.92
Range	4-11	5-10	4-8
SD	1.34	1.09	1.19
F value	0.76		
p value	0.47 (not significant)		

The onset of motor block is 6.84 min, 7.24 min and 6.92 min respectively for group I, group II and group III respectively. This is not statistically significant between groups ($p>0.05$).

Onset of motor block

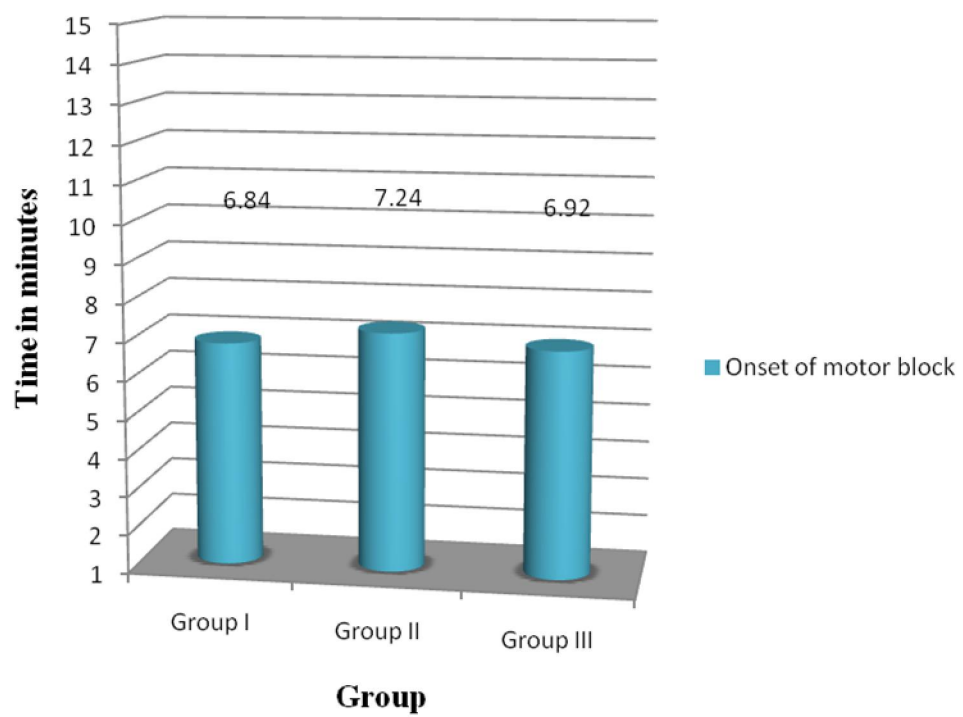


TABLE 6. DURATION OF SENSORY BLOCK

Duration of sensory block (hours)	Group I	Group II	Group III
Mean	12.96	12.68	13.24
Range	9-15	10-14	12-15
SD	1.37	0.95	0.88
F value	1.66		
p value	0.20 (not significant)		

The duration of sensory block is 12.96 hours, 12.68 hours and 13.24 hours respectively for group I, group II and group III respectively. This is not statistically significant between groups ($p>0.05$).

Duration of sensory block

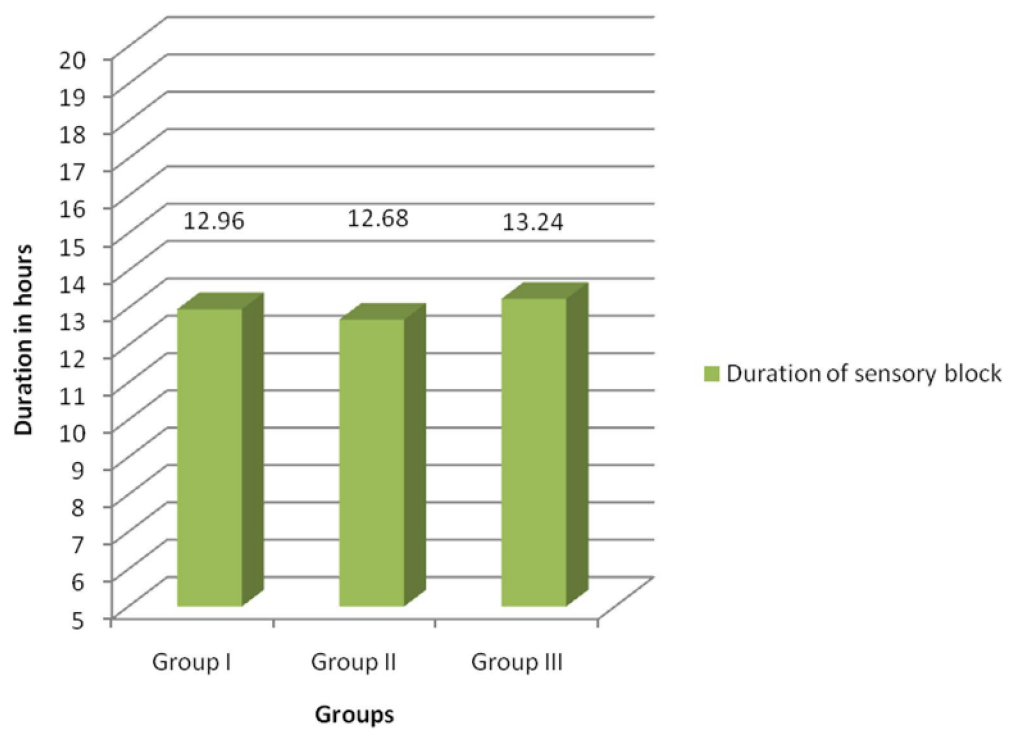


TABLE 7. DURATION OF MOTOR BLOCK

Duration of motor block (hours)	Group I	Group II	Group III
Mean	13.80	13.56	14.12
Range	11-15	9-15	13-16
SD	0.87	1.26	0.88
F value	1.90		
p value	0.16 (not significant)		

The duration of motor block is 13.80 hours, 13.56 hours and 14.12 hours respectively for group I, group II and group III respectively. This is not statistically significant between groups ($p > 0.05$).

Duration of motor block

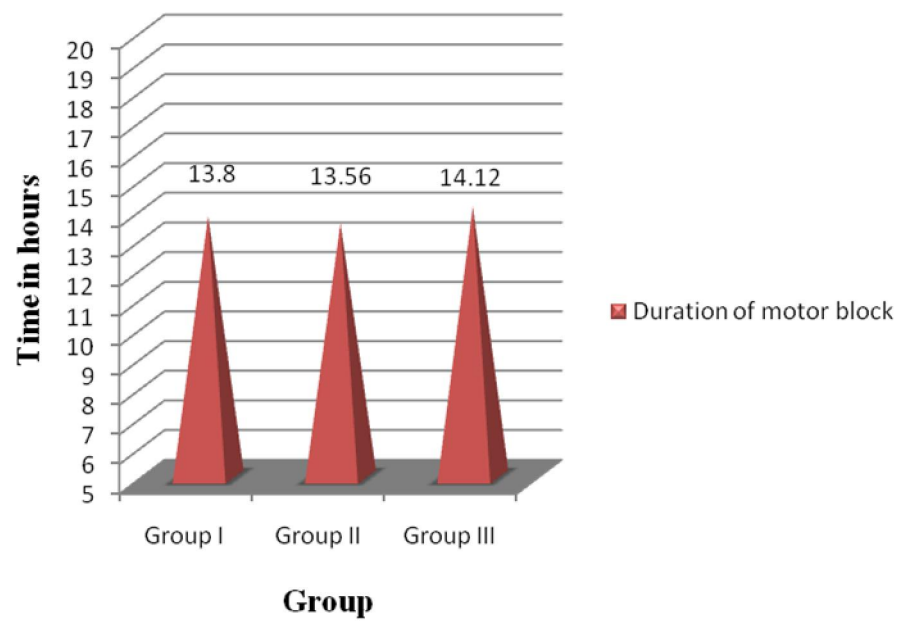


TABLE 8. HEMODYNAMIC PARAMETERS

Parameters	GROUP-I			GROUP-II			GROUP-III		
	Mean over 6 Hours	Base Line	Deviation from Base Line	Mean over 6 Hours	Base Line	Deviation from Base Line	Mean over 6 Hours	Base Line	Deviation from Base Line
Heart Rate bpm	76.44 ±4.67	79.84 ±5.99	-3.40 ±1.32	73.60 ±5.03	76.08 ±5.76	-2.48 ±0.73	75.96 ±5.82	79.04 ±6.09	-3.08 ±0.27
Systolic BP mm hg	114.8 ±6.56	118.65 ±7.27	-3.85± 0.71	114.88 ±8.02	118.24 ±8.39	-3.36 ±0.37	115.52 ±5.98	120 ±6.14	-4.48 ±0.16
Diastolic BP mm hg	73.52 ±4.63	77.12 ±5.29	-3.85 ±1.18	72 ±5.29	75.36 ±5.16	-3.36 ±0.13	71.92 ±4.42	78.08 ±4.67	-6.16 ±0.25
Mean Arterial Pressure mm hg	101.08 ±5.32	104.88 ±6.57	-3.8 ±1.25	100.48 ±6.28	104.04 ±6.79	-3.56 ±0.51	101.08 ±4.55	106.12 ±5.19	-5.04 ±0.61

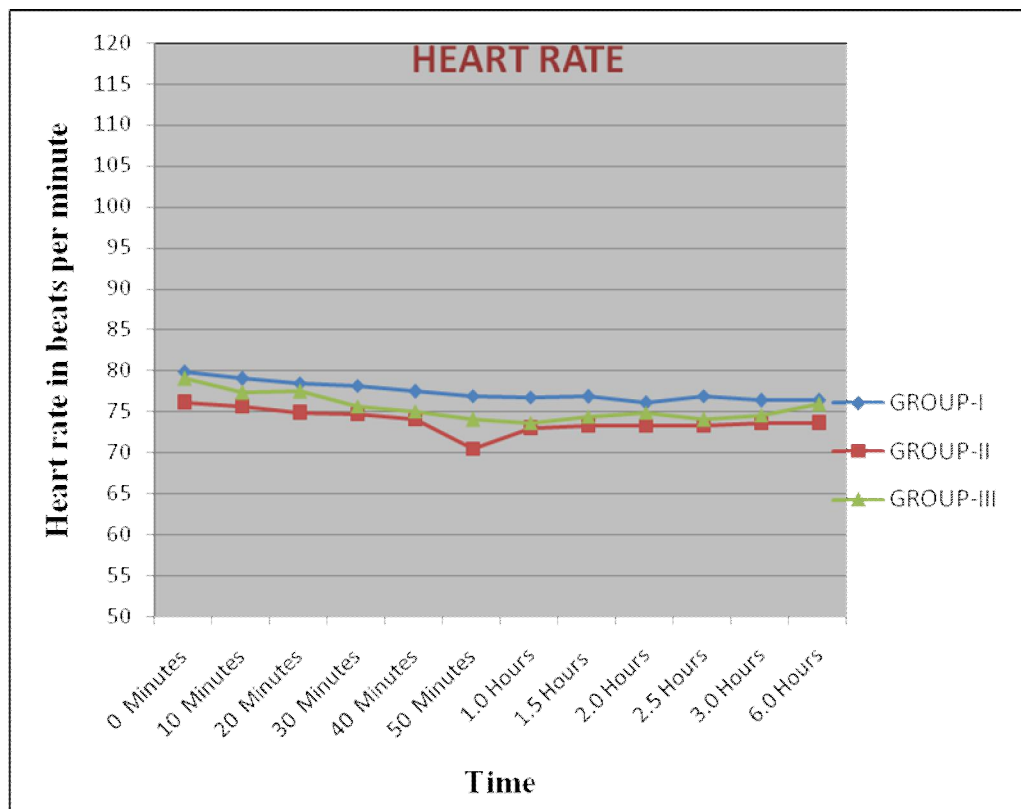
The base line and first six hours mean hemodynamic parameters (heart rate, systolic and diastolic BP and mean arterial pressure) are observed during the study period for three groups. From these the baseline value, mean value over the period of six hours and deviation from baseline (+ indicates increase, - indicates decrease) values are derived. There is no hemodynamic instability seen in three groups since the deviation from baseline is <30 %.

a) HEART RATE (bpm)

TIME	GROUP-I		GROUP-II		GROUP-III		F-Value	p value
	Mean	SD	Mean	SD	Mean	SD		
0 Minutes	79.84	6.00	76.08	5.76	79.04	6.09	2.77	0.07*
10 Minutes	79.04	6.17	75.60	6.40	77.36	6.24	1.88	0.16*
20 Minutes	78.56	6.34	74.84	4.90	77.48	5.94	4.01	0.07*
30 Minutes	78.20	5.70	74.76	4.58	75.68	6.37	3.67	0.09*
40 Minutes	77.52	5.83	74.12	5.09	75.04	6.28	3.06	0.10*
50 Minutes	77.00	6.96	70.52	14.28	74.08	5.28	2.82	0.07*
1.0 Hours	76.80	6.83	72.96	5.55	73.68	6.03	2.75	0.07*
1.5 Hours	76.88	6.61	73.28	5.07	74.44	5.09	2.66	0.08*
2.0 Hours	76.12	5.70	73.40	4.87	74.88	5.21	1.67	0.20*
2.5 Hours	76.92	5.41	73.40	4.95	74.04	6.09	2.90	0.06*
3.0 Hours	76.40	5.34	73.68	5.15	74.60	6.06	1.57	0.22*
6.0 Hours	76.44	4.67	73.60	5.03	75.96	5.82	2.14	0.13*

* Not significant

Mean heart rate is compared between three groups – baseline and then for first six hours. Statistically significant difference is not found at all time period between groups ($p > 0.05$).



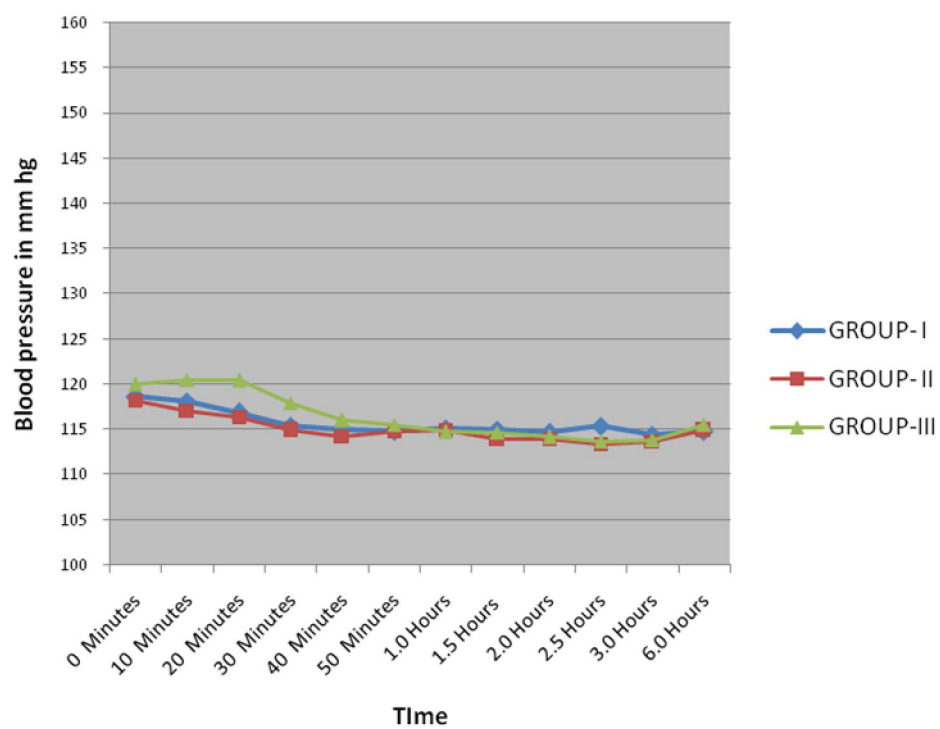
b) SYSTOLIC BLOOD PRESSURE (mm Hg)

TIME	GROUP-I		GROUP-II		GROUP-III		F-Value	p value
	Mean	SD	Mean	SD	Mean	SD		
0 Minutes	118.65	7.27	118.24	8.39	120.00	6.14	0.41	0.67*
10 Minutes	118.08	8.11	117.04	8.68	120.48	5.75	1.34	0.27*
20 Minutes	116.88	7.79	116.40	8.74	120.40	6.46	2.00	0.14*
30 Minutes	115.36	7.27	114.88	8.06	117.92	5.34	1.37	0.26*
40 Minutes	115.04	7.42	114.24	8.39	116.00	5.32	0.38	0.69*
50 Minutes	114.80	7.51	114.80	8.37	115.52	4.81	0.09	0.92*
1.0 Hours	115.12	7.12	114.96	8.61	114.72	5.13	0.02	0.98*
1.5 Hours	114.96	7.21	113.92	8.01	114.64	5.56	0.15	0.87*
2.0 Hours	114.64	7.18	113.92	8.38	114.16	5.16	0.07	0.93*
2.5 Hours	115.36	7.72	113.36	7.25	113.68	5.74	0.60	0.55*
3.0 Hours	114.40	6.90	113.68	8.44	113.84	6.11	0.07	0.93*
6.0 Hours	114.80	6.56	114.88	8.02	115.52	5.98	0.08	0.92*

* Not significant

Mean systolic blood pressure is compared between three groups – baseline and then for first six hours. Statistically significant difference is not found at all time period between groups ($p > 0.05$)

SYSTOLIC BLOOD PRESSURE



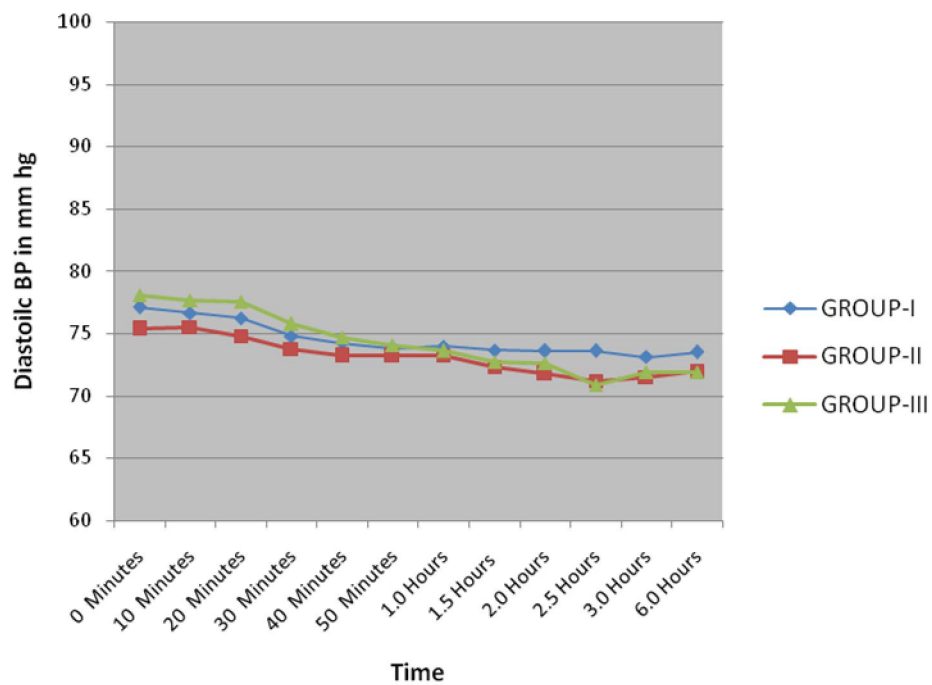
c) DIASTOLIC BLOOD PRESSURE (mm Hg)

Time	GROUP I		GROUP II		GROUP III		F-Value	p value
	Mean	SD	Mean	SD	Mean	SD		
0 Minutes	77.12	5.81	75.36	5.16	78.08	4.67	1.74	0.18 *
10 Minutes	76.64	5.91	75.52	4.87	77.68	5.09	1.04	0.36*
20 Minutes	76.24	6.04	74.80	5.42	77.60	4.32	1.74	0.18*
30 Minutes	74.80	6.66	73.76	5.52	75.76	4.29	0.81	0.45*
40 Minutes	74.24	6.17	73.20	5.42	74.72	4.32	0.53	0.59*
50 Minutes	73.84	5.68	73.20	4.83	74.08	4.64	0.20	0.82*
1.0 Hours	74.00	6.14	73.28	4.16	73.60	4.80	0.13	0.88*
1.5 Hours	73.68	5.50	72.32	5.31	72.72	4.65	0.46	0.64*
2.0 Hours	73.60	4.87	71.76	4.67	72.64	4.61	0.95	0.39*
2.5 Hours	73.60	5.20	71.20	4.24	70.88	4.48	2.55	0.09*
3.0 Hours	73.12	5.29	71.52	5.17	71.92	3.94	0.74	0.48*
6.0 Hours	73.52	4.63	72.00	5.29	71.92	4.42	0.88	0.42*

* Not significant

Mean diastolic blood pressure is compared between three groups – baseline and then for first six hours. Statistically significant difference is not found at all time period between groups ($p>0.05$)

DIASTOLIC BLOOD PRESSURE



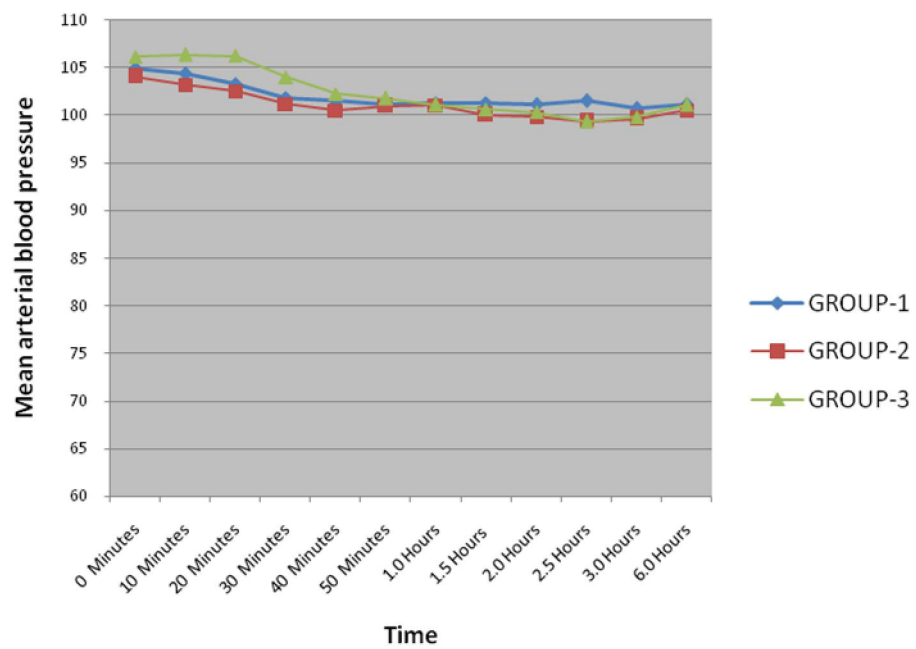
d) MEAN ARTERIAL BLOOD PRESSURE (mm Hg)

Time	GROUP I		GROUP II		GROUP III		F-Value	p value
	Mean	SD	Mean	SD	Mean	SD		
0 Minutes	104.88	6.57	104.04	6.79	106.12	5.19	0.71	0.50*
10 Minutes	104.32	7.13	103.20	7.08	106.32	4.85	1.50	0.23*
20 Minutes	103.32	7.03	102.52	7.11	106.24	4.89	2.32	0.11*
30 Minutes	101.84	6.75	101.16	6.76	103.96	4.51	1.43	0.25*
40 Minutes	101.52	6.77	100.48	6.87	102.24	4.35	0.52	0.59*
50 Minutes	101.16	6.69	100.96	6.59	101.76	4.02	0.13	0.88*
1.0 Hours	101.32	6.59	101.04	6.64	101.08	4.36	0.12	0.98*
1.5 Hours	101.32	6.44	100.00	6.64	100.64	4.64	0.31	0.74*
2.0 Hours	101.08	6.05	99.84	6.74	100.32	4.25	0.29	0.75*
2.5 Hours	101.52	6.68	99.32	5.90	99.36	4.62	1.18	0.31*
3.0 Hours	100.68	5.91	99.64	7.01	99.84	7.79	0.21	0.81*
6.0 Hours	101.08	5.32	100.48	6.28	101.08	4.55	0.10	0.90*

*Not significant

Mean MAP is compared between three groups – baseline and then for first six hours. Statistically significant difference is not found at all time period between groups ($p>0.05$)

MEAN ARTERIAL BLOOD PRESSURE



Discussion

DISCUSSION

Brachial plexus blockade offers an excellent alternative technique to general anesthesia for surgeries involving the upper limb. The advantages of regional anaesthesia over general anaesthesia are numerous and have been clearly established. It provides excellent analgesia that can be extended into the post operative period by utilizing continuous regional technique, avoidance of opioid-related side-effects (particularly nausea, vomiting and sedation), avoidance of airway instrumentation, decrease in post-operative intensity of care, decrease recovery time and improved patient satisfaction.

Ropivacaine (S-[-]-1 propyl-2', 6'pipecoloxylidide) is a new long acting anesthetic structurally closely related to bupivacaine, the difference being a propyl group instead of a butyl group linked to the piperidine ring. Contrary to racemic bupivacaine, ropivacaine is supplied as the pure S-enantiomer. It reportedly has decreased cardiovascular and central nervous system toxicity compared with bupivacaine^{42,67} and it may have clinical advantages compared with other local anesthetics. The potency of ropivacaine in terms of sensory and motor block has now been determined in clinical use. A large number of open and double blind

studies^{18,22-26,40,63,65,69-70} have been performed on human volunteer's and patients to determine the efficacy and degree of differential block with ropivacaine compared with bupivacaine in peripheral and central neural block.

The onset and duration time of ropivacaine for different regional anesthetic techniques is controversial. In studies comparing bupivacaine and ropivacaine for brachial plexus block, some studies report shorter onset time with ropivacaine compared with bupivacaine⁶⁸, where as other studies find similar^{65, 69} or longer⁷⁰ onset times of ropivacaine compared with bupivacaine for brachial plexus block.

Several techniques have been used to prolong the duration of regional anaesthesia. Besides the continuous infusion of local anaesthetics through catheters and recently opioids as adjuvants to local anaesthetic solutions, the addition of epinephrine appears to be the most widely used). The prolongation of action is generally related to local vasoconstriction which slows down the vascular reabsorption of local anaesthetics. Vasoconstriction is related to the action of epinephrine on alpha₁-type receptors.

Thus in this prospective randomized double blind study thus we compare the safety and efficacy of 0.5% Ropivacaine with adrenaline and 0.5% Ropivacaine without Adrenaline and 0.5% Bupivacaine in patients receiving subclavian perivascular brachial plexus block in seventy five patients (twenty five in each group) undergoing plastic surgery (both elective and emergency) on the forearm or hand. Demographics, onset and duration of block, hemodynamic changes and complications are observed and statistically analyzed.

We did a randomization since it eliminates the source of bias in treatments assignment and it facilitated blinding the type of treatments to the investigator and participants. This is done by shuffling envelopes (non- algorithmic randomization).

The **technique** used in this study is the **nerve locator** assisted subclavian perivascular brachial plexus block. It has been shown by Carlo D Franco et al ⁷⁴ that the subclavian perivascular technique of brachial plexus block provides a consistent, reproducible and effective anesthesia of the upper extremity and that the major reason for the success of this technique is that the local anesthetic is injected at the point where the plexus is reduced to its fewest components and size and the sheath is reduced to its smallest volume.

Elicitation of paraesthesia to confirm the needle position in nerve blocks is becoming less popular as it has problems in the form of direct neuronal damage by the advancing needle, patient discomfort and failure rates. While using nerve locator as an aid to the nerve blocks, these problems can be avoided. The nerve stimulator utilized is the Stimuplex DIG (B.Braun, Allentown PA) a unit found to be accurate in a recent comparative study ⁷⁵. Carlo D Franco et al ⁷⁴ showed in his study that it is far more important to get the desired response (i.e., flexion or extension of the fingers at an output of 0.5 to 0.7 mA) than to look for the minimum output possible in an effort to get closer to the nerve and hence we use 0.5 mA output.

In elderly patients the pharmacologic changes that occur include are changes in plasma protein binding (α_1 glycoprotein level increase and albumin decrease), changes in body content (decrease in lean body mass, increase in body fat and decrease in total body water), changes in drug metabolism (alterations in hepatic and renal clearance) and changes in pharmacodynamics. There is a decrease in the number and density of nerve fibers, a degeneration of axons, and an increase in motor unit action potentials with increased age ⁸⁶. The effect of these changes was demonstrated by Dorfman and Bosley ⁸⁷, who measured sensory and

motor conduction velocities of peripheral (median) nerves in elderly individuals and found an age-dependent decrease in conduction velocity of both motor and sensory fibers. In children immaturity of enzyme pathways and their replacement by other biochemical pathways and changes in pharmacokinetic parameters due to changes in protein binding and variation of body fluid distribution exists. Hence elderly and paediatric patients are excluded in this study.

Carlo D Franco et al⁷⁴ identified obesity as a possible factor contributing to either an incomplete or failed block. In patients with underlying pulmonary disease or in morbidly obese patients, pneumothorax and unilateral phrenic nerve block can cause severe symptoms of dyspnoea and lead to significant pulmonary compromise. Juliann T et al⁸⁹ in his study concluded that High BMI and ASA IV are independent risk factors for block failure in regional anesthesia technique. Hence we excluded obese patients and patients with severe pulmonary disease, ASA III and IV in our study. The patients on anticoagulation are excluded because if vascular puncture is encountered, the expanding hematoma could compress the airway and it will be difficult to access and maintain the airway⁷⁶.

Clinical reports have observed unexpectedly high stimulation thresholds in diabetic patients, even when ultrasound imaging shows contact of the needle with the nerve.⁷⁷ Conduction velocity in diabetics is slowed because of numerous factors⁷⁸ attributable to disrupted neuronal metabolism and reduced endoneurial blood flow.⁸⁰ Because longitudinal conduction of action potentials represents sequential excitation of segments of neuronal membrane, factors that diminish conduction velocity would also depress excitation by an external source of depolarization such as a needle electrode. Reduced excitability of peripheral nerves by depolarizing current has been confirmed in diabetics by clinical electrophysiologic examination.⁷⁹ Particularly important aspects of diabetic neuropathy are directly attributable to the effect of hyperglycemia *via* increased activity of the sorbitol pathway of glucose metabolism, which is activated in sensory neurons within hours of exposure to hyperglycemia and produces excitability deficits and sensory neuron dysfunction within 1–4 weeks.⁸¹ Because of above said factors we excluded patients with diabetes mellitus. Vasoconstrictors⁸⁸ produce a cardiac excitatory action which results in an increase in heart rate, force of contraction and stroke volume. Epinephrine may precipitate significant elevation in blood pressure (inadvertent IV injection). Hence hypertensive patients are excluded from our study.

During pregnancy increased sensitivity⁸⁴ (more rapid onset of conduction) may be present and alterations in protein binding characteristic of bupivacaine may result in increased concentrations of pharmacologically active unbound drug in the plasma⁸⁵. Hence we consider pregnancy as exclusion criteria.

The efficacy of 0.25% ropivacaine in subclavian brachial plexus block has been studied by Hickey et al⁷⁰ and it has been shown that it needed frequent supplementation with peripheral nerve block or general anesthesia in a large number of patients. Hence we did not choose 0.25% ropivacaine. In the study by Stephen M Klein et al²³ and Laura Bertini et al⁶³, increasing concentration of ropivacaine from 0.5% to 0.75% does not add benefit and it failed to improve the onset and duration of brachial plexus block. The use of increasing concentrations of local anesthetic solutions causes rapid increases in the blood serum level of the local anesthetic and the slope of the increase plays a significant role in the severity of toxic manifestations. Furthermore, the percentage that is protein-bound decreases as the blood serum level of local anesthetic increases, and this can increase the risks during an accidental injection of these higher concentrations. Hence we choose **0.5% of ropivacaine** to be compared with 0.5% of bupivacaine.

Because we consider that using different volumes of local anesthetic drugs to be tested would prejudice results, we use **similar volume (30 ml)** of 0.5% ropivacaine and 0.5% bupivacaine. Peak plasma concentration of local anesthetic depends on many variables, as well as body weight and in particular it is accepted that the maximum safe dose of local anesthetic differ according to the injection site. Thus in our study we compare equal dose and volume of ropivacaine and bupivacaine.

No significant difference in the age, weight and sex ratio is seen in our study.

There is no significant difference in onset and duration of sensory and motor block in our study between groups. This is similar to the results of the study by Hickey R et al ¹⁸ (A comparison of ropivacaine 0.5% and bupivacaine 0.5% for brachial plexus block) on 40 patients who received subclavian perivascular brachial plexus block for upper-extremity surgery. He concluded that ropivacaine 0.5% and bupivacaine 0.5% were similar in terms of onset and duration of sensory and motor block. In another study, Hickey R et al ²⁶ compared 0.5% ropivacaine with epinephrine and 0.5% ropivacaine without epinephrine in 32 patients receiving a subclavian perivascular block for upper extremity surgery. He demonstrated that ropivacaine 0.5% is an effective agent for brachial

plexus anaesthesia and the addition of epinephrine did not significantly affect the onset and duration of sensory and motor block.

A Comparison of 0.5% Bupivacaine, 0.5% Ropivacaine, and 0.75% Ropivacaine for Interscalene Brachial Plexus Block was done by Stephen M. Klein²⁵ et al in seventy-five adult patients. All solutions contained fresh epinephrine in a 1:400,000 concentration. He found there was no clinically important difference in times to onset and recovery of interscalene block. Vainionpaa et al⁶⁵ in his study compared the clinical and pharmacokinetic properties of ropivacaine and bupivacaine (both 5 mg/mL) used in axillary plexus in 60 patients. He concluded that no statistically significant differences were found between ropivacaine and bupivacaine in either the clinical or the pharmacokinetic comparisons.

The rapidity of onset of anesthesia after injection of a local anesthetic solution into tissues around a peripheral nerve depends on the pKa of the drug (The pK determines the amount of local anesthetic that exists in the active non ionized form at the pH of the tissue. pKa of ropivacaine and bupivacaine being same - 8.1)

Duration of peripheral nerve block anesthesia depends on the dose of local anesthetic, its lipid solubility, its degree of protein binding,

concentration and use of vasoconstrictor. Ropivacaine seems to increase vascular smooth muscle activity and decreases blood flow at the site of injection over a wider range of concentration compared with bupivacaine¹⁹. Kopacz et al²¹ who found that unlike bupivacaine, which produced cutaneous vasodilatation, ropivacaine produced vasoconstriction and a reduction of cutaneous blood flow in pigs. When epinephrine was added to ropivacaine no further reduction in blood flow was seen whereas when added to bupivacaine it reduced blood flow, thus showing that addition of epinephrine to ropivacaine would have less effect on its onset and duration as shown in our study.

Heart rate, systolic, diastolic and mean arterial blood pressure are monitored. No hemodynamic instability is noted in the groups after block. One patient in group I and one in group III had failed block and converted to general anesthesia. These patients' data are not included in the study. No block complication is observed in our study and no supplementation was required. Adverse event seen is nausea (one patient in group I and one in group III) and vomiting (one patient in group III). No systemic toxicity is noted.

Summary

SUMMARY

This prospective randomized double blind study compared the safety and efficacy of 0.5% ropivacaine without epinephrine, 0.5% ropivacaine with epinephrine (1:2,00,000) and 0.5% bupivacaine in patients receiving subclavian perivascular brachial plexus block using nerve locator for surgeries on forearm and hand(both elective and emergency). Seventy five patients are included in the study, they are divided in to three groups.

- i. Group I (n=25) receiving 30 ml Of 0.5% ropivacaine without epinephrine.
- ii. Group II (n=25) receiving 30 ml of 0.5% ropivacaine with epinephrine.
- iii. Group III (n=25) receiving 30 ml of 0.5% bupivacaine.

After local anesthetic injection following parameters are observed.

- i. Time of onset of sensory block.
- ii. Time of onset of motor block.
- iii. Duration of sensory block.
- iv. Duration of motor block.
- v. Vital parameters.

All the three groups are comparable in the distribution of age, sex and weight. The onset and duration of sensory block did not differ significantly between the groups. Similarly there is not statistically significant difference between the groups in onset and duration of motor block. The addition of epinephrine did not significantly affect the onset and duration of sensory and motor block of 0.5% ropivacaine.

No hemodynamic instability is observed after the block in all the groups during the study period. There is no evidence of cardiovascular or central nervous system toxicity in the study groups with the dose of local anesthetic used in our study.

Hence, Ropivacaine, a relatively new local anesthetic at a concentration of 0.5% is equally effective as 0.5% Bupivacaine. With the theoretical advantage of lesser cardiotoxicity than Bupivacaine, Ropivacaine may probably be a more safer drug than Bupivacaine in patients receiving subclavian perivascular brachial plexus block.

Conclusion

CONCLUSION

In conclusion, 0.5% ropivacaine and 0.5% bupivacaine are found to be equally efficacious in terms of onset and duration of sensory and motor block for subclavian perivascular brachial plexus block. The addition of epinephrine to ropivacaine does not reduce the onset time or prolong the duration of sensory and motor block. There is no evidence of cardiovascular or central nervous system toxicity in all groups with the dose administered in the study. Thus, the selection of the optimal long acting local anesthetic for peripheral nerve block must take into consideration of the available local anesthetics, time to onset, duration of blockade and side effects of each drug and dose. Since it has been shown that efficacy of 0.5% ropivacaine and 0.5% bupivacaine is same and ropivacaine has a theoretical advantage of lesser cardiotoxicity over bupivacaine, it may offer clinical advantage.

Annexure

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Proforma

PROFORMA

Date	:	Serial No	:
Name	:	Weight	:
Age / Sex	:		
IP Number	:		
Diagnosis	:		
Surgery planned	:		
ASA status	:		
Shifted to OT time	:	IV access	:
Monitors	:	Baseline	
Block time	:	HR	:
Surgery starting time	:	BP	:
Surgery ending time	:	SPO2:	

PARAMETERS OBSERVED:

- Onset of sensory block - _____
- Onset of motor block - _____

Time	0 m	10m	20m	30m	40m	50m	60m	1 ½ h	2 h	2 ½ h	3h	6 h	9 h	12 h	18 h	24 h
HR																
Systolic BP																
Diastolic BP																
Mean arterial BP																

Time	0 m	5 m	10 m	15 m	20 m	25 m	30 m	35 m	40 m	50 m	1 h	2 h	3 h	4 h	5 h	6 h	7 h	8 h	9 h	10 h	11 h	12 h	13 h	14 h	15 h	16 h	17 h	18 h	20 h	24 h
Duration of sensory block																														
Duration of motor block																														

Remarks if any:

Nausea

Vomiting

Shivering

Perioral numbness

Convulsions

Pruritus

Master Chart

MASTER CHART

S.number	Group	Name	Age	sex	IP number	Weight	Diagnosis	Surgery	Onset of sensory block (min)	Onset of motor block (min)	Duration of sensory block (hours)	Duration of motor block (hours)	Complication
1	I	Srinivasan	26	M	19684	62	Brachydactyl - right	Release	8	6	14	13	Nil
2	I	Venkatesh	45	M	19720	60	Post Hansen's sequelae-left	Arthodesis – PIP joint	8	8	13	15	Nil
3	I	Kumar	18	M	19658	58	Crush injury- right hand	Shortening & closure	7	7	13	14	Nil
4	II	Raganathan	34	M	19659	64	PTRA-Left hand	SSG	7	6	11	13	Nil
5	II	Sasikala	26	F	19688	54	PTRA- Right hand	Debridement	10	10	14	14	Nil
6	II	Niyamath	22	M	19642	65	PTRA-Left hand	Wound debridement	9	8	13	13	Nil
7	II	Shruthi	20	F	19777	50	Post burn scar over wrist joint	Excision	9	8	14	12	Nil
8	III	Sasikala	26	F	19758	55	PTRA- right hand	Debridement	7	5	12	14	Nil
9	I	Raviraja	27	M	19702	65	PTRA – left hand	Wound debridement	7	6	11	13	Nil
10	I	Kasi	35	M	19738	65	PTRA right thumb	SSG	8	7	15	14	Nil
11	II	Moumina	18	F	19804	60	PBSC finger- right hand	Release	7	5	12	15	Nil
12	I	Anavoo	45	M	19784	65	PTRA- right hand	SSG	8	4	14	14	Nil
13	I	Sajitha	23	F	19688	50	PTRA- right hand	SSG	9	8	15	14	Nil
14	III	Pradeep	18	M	19696	63	Raw area left hand	SSG	9	6	13	14	Nil
15	III	Muchagandhi	18	M	19724	64	PTS- right index	Rotational osteotomy	9	8	13	14	Nil
16	III	Manivannan	20	M	19920	65	PTRA- left hand	Debridement	8	8	13	13	Nil
17	II	Pradeepa	28	F	19958	63	Raw area- left hand	SSG	9	8	13	14	Nil
18	III	Karthi	21	M	19968	58	PB gangrene- left hand	Shortening and closure	9	7	13	15	Nil
19	II	Aparna	19	F	19928	60	PBSC left index finger	Release	9	8	13	14	Nil
20	III	Devaraj	20	M	19978	65	PTRA right hand	Debridement	8	8	13	13	Nil

S.number	Group	Name	Age	sex	IP number	Weight	Diagnosis	Surgery	Onset of sensory block (min)	Onset of motor block (min)	Duration of sensory block (hours)	Duration of motor block (hours)	Complication
21	III	Rajeshwari	24	F	20012	64	Crush injury right hand	Wound debridement	9	8	14	14	Nil
22	I	Praveen	18	M	20019	62	Cruch injury right hand	Wound debridement	9	7	9	11	Nil
23	II	Pikkas	22	M	20102	63	PTRA-right index finger	Debridement	8	7	12	14	Nil
24	I	Kandasamy	45	M	20137	64	Pt gangrene right index finger	Shortening and closure	8	7	13	14	Nil
25	II	Arumugam	30	M	20258	64	PTRA right hand	Wound debridement	9	8	13	14	Nil
26	III	Venkatesan	22	M	20277	65	PT gangrene right index finger	Shortening and closure	9	7	13	15	Nil
27	III	Anushya	18	F	20428	58	PTRA right hand	SSG	7	7	12	14	Nil
28	III	Raj	22	M	20297	62	Crush injury right hand	Wound debridement	8	8	13	16	Nil
29	II	Vasantha	40	F	20388	50	PTS left index finger	Sequestrectomy	8	7	12	14	Nil
30	I	Thirupathy	20	M	20327	65	Cracker burst injury rt hand	Wound debridement	7	6	13	13	Nil
31	III	Kasi	37	M	20317	62	PBSC right index finger	Release	9	8	13	15	Nil
32	II	Guhankumar	18	M	20319	64	PT gangrene right index finger	Shortening and closure	9	8	10	13	Nil
33	III	Vikkiraj	18	M	20357	50	PBSC left hand	Release	9	6	15	15	Nil
34	I	Jhansirani	36	F	20372	52	PTS right index finger	Scar excision	8	7	13	14	Nil
35	I	Shankar	25	M	20567	62	PTRA right hand	SSG	8	6	9	14	Nil
36	II	Arumugam	25	M	20569	65	PTRA right hand	Shortening and closure	9	7	13	15	Nil
37	I	Sandeepkumar	18	M	20618	50	PBSC right index finger	Release	8	7	12	15	Nil
38	III	Mariya	27	F	20717	58	Lipoma left hand	Excision	8	7	12	14	Nil
39	III	Selva kumar	20	M	20617	60	Cellulitis right hand	Wound debridement	9	7	14	14	Nil
40	III	Prabhakaran	31	M	20557	62	Crush injury left thumb	Wound debridement	9	5	14	15	Nil
41	I	Thilakaraj	23	M	20619	63	Crush injury right hand	Wound debridement	7	5	14	14	Nil

S.number	Group	Name	Age	sex	IP number	Weight	Diagnosis	Surgery	Onset of sensory block (min)	Onset of motor block (min)	Duration of sensory block (hours)	Duration of motor block (hours)	Complication
42	II	Lingamoorthy	42	M	20719	65	PTRA left hand	SSG	8	8	14	14	Nil
43	III	Boomilingam	18	M	20779	58	Syndactyl left hand	Release and SSG	7	5	12	13	Nil
44	III	Manohar	22	M	20801	58	PTRA left index finger	Ray amputation	8	6	13	13	Nil
45	I	Tamilselvi	19	F	20814	53	PBSC right hand	Release	8	11	15	14	Nil
46	II	Dineshkumar	19	M	20799	60	Scar contracture rt index finger	Excision	8	8	13	14	Nil
47	I	Nesiya	21	F	20887	52	Syndactyl right hand	Release	8	8	14	13	Nil
48	II	Divya	18	F	20981	50	Ganglioma left hand	Excision	5	6	14	15	Nil
49	III	Srinivasan	30	M	20988	65	PTRA right hand	Wound debridement	8	8	14	14	Nil
50	III	Ramesh	18	M	20908	65	PTRA left index finger	Debridement	7	4	14	13	Nil
51	II	Girish	32	M	20915	62	PTS gangrene right thumb	Wound debridement	8	5	13	14	Nil
52	I	Rajesh	19	M	20927	60	Raw area left hand	Wound debridement	15	7	13	14	Nil
53	I	Selvi	32	F	20932	56	PTRA left middle finger	Wound debridement	8	7	13	15	Nil
54	II	Prabhakaran	31	M	20944	62	PTRA left thumb	Wound debridement	8	7	12	13	Nil
55	III	Sanjay	27	M	20872	65	PTRA left hand	Wound debridement	10	8	13	14	Nil
56	III	Subramani	30	M	20919	62	PTS left hand	Scar release	8	7	14	14	Nil
57	II	Tamilselvi	29	F	20976	55	PBSC left index finger	Release	7	6	12	14	Nil
58	II	Nagaraj	35	M	20918	64	PTRA-right hand	Debridement	9	8	13	14	Nil
59	I	Kamaraj	23	M	21004	60	PTSC left index,middle&ring finger	Z plasty	8	7	13	14	Nil
60	I	Viswanathan	30	M	21017	62	Trigger finger	Release	10	7	13	13	Nil

S.number	Group	Name	Age	sex	IP number	Weight	Diagnosis	Surgery	Onset of sensory block (min)	Onset of motor block (min)	Duration of sensory block (hours)	Duration of motor block (hours)	Complication
61	III	Abdul basheer	35	M	21067	60	PTS right hand	PIP arthrodesis	9	7	14	16	Nil
62	III	Michael raj	40	M	21089	65	PTRA left hand	Debridement	9	8	13	15	Nil
63	I	Pandian	25	M	21172	65	PTRA left hand	Debridement	9	8	13	14	Nil
64	I	Om Prakash	25	M	21169	64	PTRA right hand	Wound debridement	7	5	12	13	Nil
65	II	Munirathinam	32	F	21158	61	PBSC left hand	Release	12	7	13	13	Nil
66	II	Lakshmi	39	F	21192	56	PTRA middle finger	Wound debridement	9	8	12	12	Nil
67	II	Revathy	19	F	21204	52	PTS right hand	Release	9	7	13	13	Nil
68	III	Kuppusamy	45	M	21214	60	PT amputation left middle finger	Shortening and closure	8	7	15	13	Nil
69	I	Ganesh	21	M	21219	64	Trigger thumb	Release	8	6	13	14	Nil
70	I	Anjali	25	F	21234	62	Soft tissue tumour left hand	Excision biopsy	8	6	11	15	Nil
71	I	Tamilselvan	21	M	21278	64	PBSC left finger	Release	9	8	12	14	Nil
72	III	Michealraj	26	M	21297	64	PTRA left hand	Wound debridement	9	8	12	14	Nil
73	II	Perumal	30	M	21304	65	PTRA left hand	Wound debridement	8	7	13	15	Nil
74	II	Newton	30	M	21322	65	Crush injury left hand	Wound debridement	7	7	12	9	Nil
75	II	Saravanan	30	M	21405	62	PTRA right hand	Debridement	8	7	13	14	Nil

S.NO	GROUP	PATIENT NAME	HEART RATE (bpm)												SYSTOLIC BLOOD PRESSURE (mmHg)											
			0	10	20	30	40	50	1	1.5	2	2.5	3	6	0	10	20	30	40	50	1	1.5	2	2.5	3	6
			minutes						hours						minutes						Hours					
1	I	srinivasan	70	72	68	74	66	66	65	68	68	68	65	70	110	108	104	102	104	106	108	106	108	108	110	112
2	I	Venkatesh	74	74	74	72	72	72	70	70	70	71	72	74	120	118	118	118	116	118	120	122	124	126	118	120
3	I	Kumar.s	88	88	88	86	87	89	85	86	84	84	83	84	128	124	120	116	116	118	116	116	116	116	118	118
4	II	Ranganathan	70	70	72	70	72	70	70	68	68	70	66	70	126	124	124	124	126	126	128	120	120	120	126	122
5	II	sasikala	74	76	74	74	72	74	70	70	70	72	70	72	110	108	108	108	110	110	110	106	108	106	106	110
6	II	Niyamath	68	64	68	70	66	68	70	70	70	72	68	66	120	118	120	116	116	118	120	122	128	124	124	128
7	II	shruthi	70	70	70	70	68	68	66	68	68	70	68	66	106	106	104	104	102	102	104	104	102	106	102	108
8	III	Sasikala	88	86	84	82	80	80	84	82	82	82	84	80	110	110	108	110	110	118	108	106	106	108	108	106
9	I	raviraja	86	84	82	82	84	86	84	82	78	80	80	84	120	120	118	118	118	118	120	116	116	118	118	116
10	I	kasi	72	72	72	74	70	68	68	66	70	75	76	74	120	120	120	118	118	118	118	120	122	122	118	118
11	II	Moumina	76	76	74	74	72	70	68	72	72	70	74	72	110	110	108	106	110	112	110	116	106	108	106	108
12	I	anavoo	80	82	80	76	74	76	76	74	74	78	75	78	128	130	130	128	126	126	126	126	126	128	126	126
13	I	sajitha	86	82	78	78	78	80	80	78	76	74	76	75	110	110	110	108	108	108	106	106	106	110	106	108
14	III	Pradeep	78	78	78	74	74	72	70	74	76	72	70	76	120	122	118	120	118	116	118	116	114	114	116	114
15	III	Muchagandhi	80	76	80	80	80	74	72	74	74	72	76	78	130	130	130	128	126	126	124	124	124	122	120	126
16	III	Manivannan	78	78	78	76	74	72	72	72	70	70	74	72	120	120	118	116	118	116	114	116	116	114	114	114
17	II	Pradeepa	80	82	78	78	76	78	72	76	76	76	78	72	120	120	116	116	114	118	114	110	110	114	116	108
18	III	Karthik	80	80	80	80	78	76	76	74	72	74	76	78	126	120	124	126	116	118	118	122	118	118	124	120
19	II	aparna	72	70	72	72	70	68	69	70	66	65	70	72	110	108	108	106	104	104	108	106	108	108	104	112
20	III	Devaraj	80	80	80	80	78	76	76	76	74	74	74	76	122	124	120	120	118	116	116	118	118	120	116	118
21	III	Rajeswari	88	88	88	86	86	86	84	84	82	86	84	86	120	122	118	116	114	116	118	114	112	112	110	114
22	I	praveen	78	76	78	72	72	72	70	70	72	74	76	74	120	120	118	118	116	116	116	114	114	114	110	110
23	II	pikkas	72	70	70	68	70	68	66	66	68	64	68	68	116	116	118	110	116	116	114	108	108	112	110	116
24	I	kandasamy	76	74	72	72	70	66	70	70	74	72	72	72	130	134	128	122	120	120	124	122	120	122	122	122
25	II	arumugam	78	72	70	72	70	71	71	74	72	74	70	72	130	130	128	122	120	120	122	124	126	126	122	122
26	III	Venkateshan	72	72	71	70	70	70	72	68	68	66	66	65	120	120	124	118	118	116	118	112	112	114	110	118

S.NO	GROUP	NAME	HEART RATE (bpm)												SYSTOLIC BLOOD PRESSUR (mm HG)											
			0	10	20	30	40	50	1	1.5	2	2.5	3	6	0	10	20	30	40	50	1	1.5	2	2.5	3	6
			minutes						hours						minutes						Hours					
27	III	Anushya	88	88	86	86	84	80	80	80	82	80	78	82	126	126	124	122	118	118	122	120	120	118	120	122
28	III	Raj	80	76	76	76	72	72	74	74	75	72	76	76	120	122	118	116	118	116	114	114	116	112	112	118
29	II	vasantha	70	72	72	70	68	68	68	66	66	67	70	72	110	108	106	112	104	108	108	110	114	112	116	118
30	I	tirupathy	78	78	76	77	76	76	76	74	74	76	76	74	108	108	108	106	106	104	106	106	110	110	108	108
31	III	Kasi	70	70	70	68	68	66	66	65	68	66	66	72	110	110	116	108	106	108	106	106	108	104	108	106
32	II	guhankumar	80	76	76	76	74	76	74	72	76	76	76	74	120	118	118	116	120	116	118	120	120	116	116	110
33	III	Vikkiraj	76	72	72	72	70	70	68	74	78	70	72	74	110	116	110	112	108	106	104	104	106	106	110	112
34	I	jhansirani	84	84	80	82	83	86	88	84	82	80	78	78	118	116	116	116	118	118	120	120	116	118	118	114
35	I	Shankar	86	84	82	80	84	82	82	86	86	84	84	82	110	108	108	108	106	106	108	108	110	112	112	112
36	I	arumugam	78	74	74	76	72	72	72	74	74	75	76	76	130	126	128	126	130	132	128	128	128	126	126	128
37	I	sandeepkumar	84	82	84	82	82	78	78	78	76	78	78	76	120	120	116	116	120	122	122	122	118	118	116	116
38	III	Mariya	70	68	72	68	64	66	66	68	70	64	66	70	122	118	120	122	118	116	116	118	114	114	118	120
39	III	selvakumar	88	84	84	80	78	78	76	78	80	82	78	76	130	130	136	128	120	122	118	122	122	124	124	120
40	III	prabhakaran	86	84	84	86	82	82	80	80	76	80	82	86	122	120	126	116	118	116	114	118	114	116	116	118
41	I	thilakaraj	86	84	84	84	82	84	82	82	82	84	84	80	126	126	124	124	122	122	122	122	120	120	122	124
42	II	lingamoorthy	70	72	72	72	74	70	70	68	72	68	70	70	110	106	108	106	104	106	106	110	110	106	108	108
43	III	boomilingam	76	72	72	70	68	74	66	74	72	70	74	74	120	120	126	118	118	116	114	114	112	116	116	114
44	III	manohar	84	82	82	84	86	80	81	82	82	78	78	82	124	126	122	122	126	122	118	114	116	118	120	120
45	I	tamilselvi	70	68	68	70	72	68	66	66	68	70	72	72	110	110	110	110	108	108	106	106	110	104	106	108
46	II	dineshkumar	70	74	76	74	76	76	76	75	78	78	78	74	110	106	108	106	106	108	108	104	106	104	106	108
47	I	Nesiya	74	72	70	70	70	68	72	74	74	73	72	74	110	110	106	106	104	106	108	108	106	104	104	104
48	II	Divya	76	78	74	74	72	70	74	73	76	76	72	70	110	110	108	106	108	108	104	106	106	104	104	106
49	III	srinivasan	70	72	68	66	68	66	67	70	72	68	64	68	110	116	108	108	104	106	106	110	108	108	104	104
50	III	Ramesh	80	80	80	76	78	76	78	75	78	80	82	78	118	116	116	118	114	114	112	114	114	116	110	112
51	II	Girish	76	74	74	74	74	72	70	74	74	72	70	68	108	106	104	106	104	104	106	106	108	108	106	106
52	I	Rajesh	76	72	74	74	76	75	72	74	74	78	69	68	110	108	108	106	108	104	104	106	104	102	100	102

S.NO	GROUP	NAME	HEART RATE (bpm)												SYSTOLIC BLOOD PRESSURE (mm HG)											
			0	10	20	30	40	50	1	1.5	2	2.5	3	6	0	10	20	30	40	50	1	1.5	2	2.5	3	6
			minutes						hours						minutes						Hours					
53	I	Selvi	80	82	80	84	82	78	78	78	76	78	76	78	120	122	126	118	118	116	114	110	106	106	108	108
54	II	prabhakaran	80	80	78	76	76	74	82	82	76	76	78	80	120	122	120	118	116	118	116	110	106	106	108	108
55	III	Sanjay	80	76	82	72	74	76	76	78	80	82	76	76	128	128	126	116	120	118	118	120	122	122	124	122
56	III	subramani	82	82	80	78	76	76	74	72	78	74	76	80	124	128	124	122	118	116	116	118	114	112	114	122
57	II	tamilselvi	80	82	80	78	78	76	76	78	76	75	76	78	118	116	114	116	112	110	108	106	108	106	108	110
58	II	Nagaraj	88	86	84	86	87	82	82	80	80	82	84	84	130	128	128	126	124	126	122	124	126	124	130	128
59	I	kamaraj	88	88	86	84	82	78	78	78	74	78	78	80	120	120	118	116	114	110	110	108	112	118	118	120
60	I	viswanathan	84	84	86	84	80	82	78	76	76	82	86	84	130	128	130	130	130	128	126	124	126	126	120	120
61	III	abdul basheer	70	68	68	66	66	70	68	69	68	67	66	68	116	116	118	114	112	110	116	110	112	108	108	116
62	III	michael raj	76	70	72	70	74	74	70	68	70	72	73	70	110	110	116	116	110	108	106	104	104	102	102	104
63	I	Pandian	70	72	74	70	72	68	68	72	66	64	68	68	118	116	116	114	112	110	114	114	110	112	112	114
64	I	Om prakash	88	90	92	88	84	84	82	82	80	78	76	76	130	132	126	118	120	116	116	126	124	126	124	126
65	II	munirathinam	80	76	76	76	78	76	74	74	72	74	75	74	126	126	128	124	122	122	124	120	122	120	124	126
66	II	lakshmi	74	72	74	74	75	70	74	78	76	78	82	80	120	120	122	120	118	118	120	116	118	114	114	116
67	II	revathy	70	72	71	71	69	67	66	66	68	70	68	74	110	110	108	106	104	102	100	104	102	110	104	104
68	III	kuppusamy	72	70	70	70	68	66	64	70	65	70	70	70	120	120	124	120	118	116	116	118	118	116	114	118
69	I	ganesh	80	82	84	86	84	85	88	90	85	86	84	82	120	120	118	122	126	128	124	122	124	122	122	118
70	I	anjali	78	74	76	76	78	80	82	80	78	76	74	76	110	106	106	106	104	106	106	108	104	106	108	110
71	I	tamilselvan	80	76	76	78	78	78	82	84	86	82	80	78	118	118	120	120	118	118	118	116	114	116	116	116
72	III	michael raj	84	82	80	76	80	74	82	80	80	80	84	86	122	122	120	116	116	118	118	114	114	108	108	110
73	II	perumal	88	92	86	84	82	86	86	84	86	85	84	86	126	124	122	118	116	118	124	122	118	118	118	116
74	II	newton	76	72	70	76	78	7	74	74	75	72	75	74	130	130	128	128	126	124	126	120	118	116	118	120
75	II	saravanan	86	88	86	84	84	86	84	80	80	78	76	76	130	130	126	126	124	124	126	126	122	120	120	126

S.NO	GROUP	NAME	DIASTOLIC BLOOD PRESSURE - mmHg												MEAN ARTERIAL PRESSURE –mmHg											
			0	10	20	30	40	50	1	1.5	2	2.5	3	6	0	10	20	30	40	50	1	1.5	2	2.5	3	6
			minute						hours						minutes						Hours					
1	I	Srinivasan	70	68	68	64	64	68	68	66	68	66	70	72	83	81	80	77	77	81	81	79	81	80	83	85
2	I	Venkatesh	80	80	80	78	78	78	82	82	82	80	82	80	93	93	93	91	91	91	95	95	96	95	94	93
3	I	Kumar.s	88	80	80	74	72	70	70	70	70	70	74	72	101	95	93	88	87	86	85	85	85	85	89	87
4	II	Ranganathan	84	82	80	80	82	80	82	82	80	80	86	82	98	96	95	95	97	95	97	95	93	93	99	95
5	II	Sasikala	70	72	70	72	74	70	70	68	68	68	70	72	83	84	83	84	86	83	83	81	81	81	82	85
6	II	Niyamath	80	82	82	86	80	82	80	82	80	76	76	78	93	94	95	96	92	94	93	95	96	92	92	95
7	II	Shruthi	70	70	68	68	66	66	68	68	70	66	68	66	82	82	80	80	78	78	80	80	81	79	79	80
8	III	Sasikala	70	72	74	70	70	68	68	68	68	72	70	66	83	85	85	83	83	85	81	81	81	84	83	79
9	I	Raviraja	80	80	76	76	78	74	70	72	70	72	72	70	93	93	90	90	91	89	87	87	85	87	87	85
10	I	Kasi	80	80	82	80	78	78	76	78	80	80	80	76	93	93	95	93	91	91	90	92	94	94	93	90
11	II	Moumina	70	70	68	68	72	70	70	72	70	68	66	72	83	83	81	81	85	84	83	87	82	81	79	84
12	I	Anavoo	78	78	80	80	78	78	78	80	82	80	78	76	95	95	97	96	94	94	94	95	97	96	94	93
13	I	Sajitha	70	70	70	68	68	70	68	70	70	70	68	68	83	83	83	81	81	83	81	82	82	83	81	81
14	III	Pradeep	80	82	82	80	80	76	74	76	78	76	72	74	93	95	94	93	93	89	89	89	90	89	87	87
15	III	Muchagandhi	72	70	72	74	72	68	66	64	66	62	66	64	91	90	91	92	90	87	85	84	85	82	84	85
16	III	Manivanan	80	80	78	78	78	76	76	74	74	74	72	70	93	93	91	91	91	89	89	88	88	87	86	85
17	II	Pradeepa	80	80	76	76	78	76	74	72	72	70	70	68	93	93	89	89	90	90	87	85	85	85	85	81
18	III	Karthik	86	86	84	84	80	84	84	86	82	82	80	80	99	97	97	98	92	95	95	98	94	94	95	93
19	II	Aparna	70	70	70	70	68	68	68	66	66	68	68	70	83	83	83	82	80	80	81	79	80	81	80	84
20	III	Devaraj	82	82	80	80	78	78	76	72	72	70	76	78	95	96	93	93	91	91	89	87	87	87	89	91
21	III	Rajeshwari	80	80	82	76	76	78	78	76	70	72	74	74	93	94	94	89	89	91	91	89	84	85	86	87
22	I	Praveen	80	82	80	86	76	80	78	76	76	76	70	72	93	95	93	97	89	92	91	89	89	89	83	85
23	II	Pikkas	74	72	72	72	70	72	70	68	70	70	68	72	88	87	87	85	85	87	85	81	83	84	82	87
24	I	Kandasamy	80	84	80	70	70	70	74	70	70	72	76	76	97	101	96	87	87	87	91	87	87	89	91	91
25	II	Arumugam	80	80	78	76	70	70	72	74	76	76	72	74	97	97	95	91	87	87	89	91	93	93	89	90
26	III	Venkateshan	80	80	80	76	78	76	76	70	76	70	72	70	93	93	95	90	91	89	90	84	88	85	85	86
27	III	Anushya	82	82	82	80	78	78	76	76	78	72	70	70	97	97	96	94	91	91	91	91	92	87	87	87
28	III	Raj	80	80	80	80	82	78	78	76	76	72	72	78	93	94	93	92	94	91	90	89	89	85	85	91
29	II	Vasanth	70	72	70	68	68	70	72	68	66	70	72	68	83	84	82	83	80	83	84	82	82	84	87	85
30	I	Tirupathy	70	70	70	64	64	64	62	66	70	72	68	68	83	83	83	78	78	77	77	79	83	85	81	81
31	III	Kasi	70	72	72	74	72	70	68	68	68	64	64	66	83	85	87	85	83	83	81	81	81	77	79	79
32	II	Guhankumar	80	82	78	74	78	78	80	80	80	76	76	72	93	94	91	88	92	91	93	93	93	89	89	85
33	III	Vikkiraj	72	70	76	70	72	72	68	70	68	70	70	68	85	85	87	84	84	83	80	81	81	82	83	83
34	I	Jhansirani	80	78	78	78	80	76	82	80	78	80	80	86	93	91	91	91	93	90	95	93	91	93	93	95
35	I	Shankar	70	68	68	64	64	68	70	70	70	72	72	72	83	81	81	79	78	81	83	83	83	85	85	85

S NO	GROUP	NAME	DISTOLIC BLOOD PRESSURE – mm HG												MEAN ARTERIAL PRESSURE – mm HG											
			0	10	20	30	40	50	1	1.5	2	2.5	3	6	0	10	20	30	40	50	1	1.5	2	2.5	3	6
			minutes						hours						minutes						Hours					
36	II	Arumugam	80	78	78	74	72	70	72	72	74	74	74	72	97	94	95	91	91	91	91	91	92	91	91	91
37	I	Sandeepkumar	80	80	74	74	82	84	82	82	78	78	72	72	93	93	88	88	95	97	95	95	91	91	87	87
38	III	Mariya	82	82	80	80	78	76	78	76	72	70	74	80	95	94	93	94	91	89	91	90	86	85	89	93
39	III	Selvakumar	82	82	80	76	80	80	76	76	78	76	78	70	98	98	99	93	93	94	90	91	93	92	93	87
40	III	Prabhakaran	78	74	74	72	70	70	74	72	76	72	70	72	93	89	91	87	86	85	87	87	89	87	85	87
41	I	Thilakaraj	82	82	86	82	80	80	78	78	74	74	68	70	97	97	99	96	94	94	93	93	89	89	86	88
42	II	Lingamoorthy	70	74	72	72	70	72	74	76	72	70	68	68	83	85	84	83	81	83	85	87	85	82	81	81
43	III	Boomilingam	82	80	80	78	76	78	74	74	72	76	74	76	95	93	95	91	90	91	87	87	85	89	88	89
44	III	Manohar	82	80	82	78	76	76	78	74	78	74	78	74	96	95	95	93	93	91	91	87	91	89	92	89
45	I	Tamilselvi	70	70	70	70	68	68	66	68	70	68	66	70	83	83	83	83	81	81	79	81	83	80	79	83
46	II	Dineshkumar	70	68	68	68	70	72	72	70	68	66	64	62	83	81	81	81	82	84	84	81	81	79	78	77
47	I	Nesiya	70	70	68	72	72	72	70	74	72	70	72	70	83	83	81	83	83	83	83	85	83	81	83	81
48	II	Divya	70	70	68	66	68	66	72	70	70	70	72	74	83	83	81	79	81	80	83	82	82	81	83	85
49	III	Srinivasan	70	70	72	68	68	70	70	72	68	64	68	68	83	85	84	81	80	82	82	85	81	79	80	80
50	III	Ramesh	80	76	76	78	72	72	74	76	76	70	72	72	93	89	89	91	86	86	87	89	89	85	85	85
51	II	Girish	76	74	78	74	72	76	70	70	72	72	70	76	87	85	87	85	83	85	82	82	84	84	82	86
52	I	Rajesh	70	68	68	66	68	66	66	64	70	66	70	68	83	81	81	79	81	79	79	78	81	78	80	79
53	I	Selvi	78	80	80	80	78	76	72	70	70	68	70	70	92	94	95	93	91	89	86	83	82	81	83	83
54	II	Prabhakaran	80	80	86	82	78	78	76	70	72	74	70	70	93	94	97	94	91	91	89	83	83	85	83	83
55	III	Sanjay	78	78	76	76	70	68	66	70	72	72	70	68	95	95	93	89	87	85	83	87	89	89	88	86
56	III	Subramani	80	86	84	80	80	76	78	78	74	70	74	72	95	100	97	94	93	89	91	91	87	84	87	89
57	II	Tamilselvi	78	76	74	70	68	70	72	64	68	64	70	70	91	89	87	85	83	83	84	78	81	78	83	83
58	II	Nagaraj	80	82	84	84	80	80	78	80	76	72	80	78	97	97	99	98	95	95	93	95	93	89	97	95
59	I	Kamaraj	76	76	74	74	72	70	72	74	76	78	78	78	91	91	89	88	86	83	85	85	88	91	91	92
60	I	Viswanathan	82	80	84	84	84	80	86	76	74	74	70	72	98	96	99	99	99	96	99	92	91	91	87	88
61	III	abdul basher	72	70	72	70	70	70	68	66	68	70	76	76	87	85	87	85	84	83	84	81	83	83	87	89
62	III	michael raj	76	76	72	70	72	70	70	68	68	64	68	70	87	87	87	85	85	83	82	80	80	77	79	81
63	I	Pandian	76	74	72	70	72	70	74	74	72	74	74	76	90	88	87	85	85	83	87	87	85	87	87	89
64	I	Om prakash	90	90	86	80	80	76	78	80	82	84	84	82	103	104	99	93	93	89	91	95	96	98	97	97
65	II	Munirathinam	80	80	80	82	86	80	74	76	74	76	80	82	95	95	96	96	98	94	91	91	90	91	95	97
66	II	Lakshmi	70	72	74	70	68	68	72	68	68	70	70	72	87	88	90	87	85	85	88	84	85	85	85	87
67	II	Revathy	70	70	68	68	66	68	66	66	62	64	64	64	83	83	81	81	79	79	77	79	75	79	77	77
68	III	Kuppusamy	74	72	70	70	68	66	68	68	64	66	66	68	89	88	88	87	85	83	84	85	82	83	82	85
69	I	Ganesh	80	80	82	84	84	84	82	80	78	76	76	74	93	93	94	97	98	99	96	94	93	91	91	89
70	I	Anjali	70	70	70	72	70	68	70	66	64	64	62	70	83	82	82	83	81	81	82	80	77	78	77	83

S. NO	GROUP	NAME	DISTOLIC BLOOD PRESSURE- mm HG												MEAN ARTERIAL BLOOD PRESSURE – mm HG											
			0	10	20	30	40	50	1	1.5	2	2.5	3	6	0	10	20	30	40	50	1	1.5	2	2.5	3	6
			minutes						hours						minutes						Hours					
			78	78	80	80	76	78	76	76	74	76	76	78	91	91	93	93	90	91	90	89	87	89	89	91
71	I	Tamilselvan	78	78	80	80	76	78	76	76	74	76	76	78	91	91	93	93	90	91	90	89	87	89	89	91
72	III	michael raj	82	80	80	76	72	78	78	72	74	72	72	74	95	94	93	89	87	91	91	86	87	84	84	86
73	II	Perumal	82	80	78	76	74	76	80	80	78	78	76	78	97	95	93	90	88	90	95	94	91	91	90	91
74	II	Newton	80	80	78	76	76	74	72	70	70	72	70	76	97	97	95	93	93	91	90	87	86	87	86	91
75	II	Saravanan	70	72	72	72	76	78	76	76	72	70	68	64	90	91	90	90	92	93	93	93	89	87	85	85

Informed Consent

சுய ஒப்புதல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு :

சங்கிளேவியன் பெரிவாஸ்கலர் பிரேக்கியல் பக்கலஸ் பிளாக்கின் 0.5%
புபிவேகன் 0.5% ரோபிவேகேயன் 0.5% ரோபிவாகேன் உடன்
1 : 2,00,000 அடரினலினும் மயக்க மருந்துகளை ஒப்பிடும் சோதனை

ஆராய்ச்சி நிலையம் :

அரசு ஸ்டான்லி மருத்துவமனை
சென்னை - 600 001.

நோயாளியின் பெயர் :

வயது :

நோயாளியின் எண் :

பாலினம் : ஆண் ☐ பெண் ☐

விலாசம் :

பங்குபெறும் நபர்கள் () குறிக்கவும்

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும். அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது.

☐

நான் இவ்வாய்வில் தன்னிச்சையாகதான் பங்கேற்க வைக்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும் எந்த சட்ட சிக்கலுக்கும் உட்படாமல் என் இவ்வாய்வில் இருந்து விலக்கி கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.

☐

இந்த ஆய்வு சம்பந்தமாகவோ, அதை சார்ந்த மேலும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் எனக்கு மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். என் ஆய்வில் இருந்து விலக்கி கொண்டாலும் இது பொருந்தும் என அறிகிறேன்.

☐

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும், பரிசோதனை முடிவுகளையும் மற்றும் சிகிச்சை தொடர்பான தகவல்களையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக் கொள்ளவும் அதை பிரசுரிக்கவும் என் முழு மனதுடன் சம்மதிக்கிறேன்.

☐

இந்த ஆய்வில் என்னை ஈடுபடுத்த முழுமனதுடன் ஒப்புக் கொள்கிறேன். இந்த மயக்க மருந்துகள் மற்றும் மயக்க முறையினால் ஏற்படக் கூடிய பின் விளைவுள் மற்றும் எதிர்பாராத விளைவுகள் பற்றி எனக்கு விளக்கமாக தெரிவிக்கப்பட்டது.

☐

இந்த ஆய்வில் எனக்கு கழுத்தில் ஊசி போட்டு மயக்கம் கொடுத்த பின் ரோபிவேகேயன், ரோபிவேகேயன் அடரினலின் அல்லது புபிவேகேயன் கொடுக்க சம்மதிக்கிறேன். மேலும், அறுவை சிகிச்சைக்குப் பின் எனக்கு வலி, உணர்ச்சியின் அளவு போன்ற அனைத்து விதமான பரிசோதனைகளையும் செய்து பார்க்க நான் முழுமனதுடன் சம்மதிக்கிறேன்.

☐

நோயாளின் கையொப்பம்..... இடம் தேதி

கட்டைவிரல் ரேகை

ஆய்வாளரின் கையொப்பம் இடம் தேதி

ஆய்வாளரின் பெயர் :

சங்கிலேவியன் பெரிவாஸ்கலர் பிரேக்கியல் பக்கலஸ் பிளாக்கின் 0.5%
புபிவேகன் 0.5% ரோபிவேகேயன் 0.5% ரோபிவாகேன் உடன்
1 : 2,00,000 அடரினலினும் மயக்க மருந்துகளை ஒப்பிடும் சோதனை
நோயாளிகளுக்கான தகவல் :

ஆராய்ச்சியின் நோக்கமும், ஆதாயங்களும் :

உங்களை ஈடுபடுத்த திடமிடப்பட்டுள்ள இந்த மருத்துவ ஆராய்ச்சி ஆய்வானது சங்கிலேவியன் பெரிவாஸ்கலர் பிரேக்கியல் பக்கலஸ் பிளாக் 0.5% புபிவேகன் 0.5% ரோபிவேகேயன் 0.5% ரோபிவேகேயன் உடன் 1 : 2,00,000 அடரினலினும் மயக்க மருந்துகளை ஒப்பிடும் சோதனை. அதனால் ஏற்படும் வலி நிவாரணம் போன்ற குறியீடுகளை ஒப்பிட்டுப் பார்ப்பதை நோக்கமாக கொண்டுள்ளதாகும். மருத்துவ விவரக் கையேட்டில் அறிவிக்கப்பட்டுள்ள ஆய்வுகளின் படி, ரோபிவேகேயன் மருந்து புபிவேகேயன் மருந்தைவிட குறைவான இருதய பாதிப்பையே ஏற்படுத்துகிறது. இந்த மருந்து பாதுகாப்பானதாகவும் இருப்பதாக அறியப்படுகிறது. இம்மருந்தினால் நீங்கள் பயனடைவீர்கள் என எதிர்பார்க்கப்படுகிறது. இந்த ஆய்வின் மூலம் பெறப்படும் அறியவானது, இது போன்ற அறுவை சிகிச்சை செய்து கொள்ளும் மற்றவர்களுக்கும் பயனுடையதாக அமையும்.

மாற்று மயக்க மருந்துகள்.

சங்கிலேவியன் பெரிவாஸ்கலர் பிரேக்கியல் பக்கலஸ் பிளாக் ரோபிவேகேயன் மற்றும் புபிவேகேயன் மருந்துகள் அல்லாமல் லிக்னோகேயன் போன்ற மருந்துகளும் உபயோகத்தில் உள்ளன. இது மேற்குறிப்பிட்டுள்ள இரண்டு மருந்துகளை விட குறைவான நேரமே வலி நிவாரணம் அளிக்கும் மாற்று மயக்க முறைகள் இது போன்ற முழங்கை கீழே செய்யப்படும் அறுவை சிகிச்சைக்கு வெறும் முழு மயக்கமே முன்னால் கொடுக்கப்பட்டு வந்தது. இந்த முறையில் வலி நிவாரணிகளை இரத்த நாளத்தின் மூலமாக கொடுப்பதினால் ஏற்படக் கூடிய மூச்சு விடுவதில் சிரமம் மற்றும் போதிய வலி நிவாரணம் இல்லாதது போன்ற சிரமங்கள் உள்ளன.

உண்டாகக் கூடிய இடர்கள்

அனைத்து மயக்க மருந்து மற்றும் மயக்க முறைகளுடன் இருப்பது போலவே இந்த முறையிலும் சில எதிர்பாராத இடர்கள் நடைபெறலாம். சங்கிலேவியன் பெரிவாஸ்கலர் பிரேக்கியல் பக்கலஸ் பிளாக் முறையில் மிக அரிதாக இந்த மயக்க மருந்துகள் இரத்தத்திலோ அல்லது நுரையரலில் பாதிப்போ செலுத்தப்படுவதற்கான வாய்ப்பு உள்ளன. அப்படியாக நேரும் பட்சத்தில், அது இருதய பாதிப்பையும் அல்லது மூளை பாதிப்பையும் ஏற்படுத்தலாம். இந்த மருந்துகளின் ஒரு சில பேருக்கு அறுவைசிகிச்சைக்குப் பின் வாந்தி போன்ற சிறு பிரச்சனைகள் ஏற்பட வாய்ப்புள்ளது.

இந்த ஊறுகளை குறைக்கும் பொருட்டு, மருத்துவரால் செய்யப்படும் பொருத்தமான கூர்ந்தாராய்வு சோதனைகள் மற்றும் முன்னெச்சரிக்கை நடவடிக்கைகளுடன் இந்த ஆய்வை வடிவமைக்க அக்கறை எடுத்துக் கொள்ளப்பட்டுள்ளது. ஆய்வு பொறுப்பேற்றுள்ள மருத்துவர், இந்த ஆய்வு மருந்துகளுடன் தொடர்புடைய மற்ற

அனைத்துப் பிற இடர்களைப் பற்றியும் உங்களுக்கு விளக்குவார்.

ஆய்வு நடைமுறைகள்

சிறிய ஊசியின் மூலம் கழுத்தில் உள்ள நரம்புகள் செல்லும் பாதையில் மயக்க மருந்தினை செலுத்தி அதனால் ஏற்படும் வலி நிவாரணம் கொடுக்கப்படும். அறுவை சிகிச்சைக்குப்பின் உங்களின் வலி, உணர்ச்சியின் அளவு மற்றும் தசைகளை இயக்கும் திறன் ஆகியவை பரிசோதிக்கப்படும். உங்களுக்கு வலி ஏற்பட்டால் அதற்கான வலி நிவாரணி கொடுக்கப்பட்டு தொடர்ந்து நீங்கள் மருத்துவரால் கண்காணிக்கப்படுவீர்கள்.

ஆய்வில் உங்கள் உரிமைகள்

உங்கள் மருத்துவப் பதிவேடுகள் மிகவும் அந்தரங்கமாக வைத்துக் கொள்ளப்படும். இந்த ஆய்வின் முடிவுகள் அறிவியல் பத்திரிக்கைகளில் பிரசுரிக்கப்படலாம். ஆனால், பெயரை வெளியிடுவது மூலம் அடையாளம் காட்டப்படமாட்டார்கள். இந்த ஆய்வில் உங்கள் பங்கேற்பு தன்னிச்சையானது மற்றும் காரணங்கள் எதையும் கூறாமலேயே நீங்கள் இந்த ஆய்விலிருந்து எந்த ஒரு நேரத்திலும் விலகிக் கொள்ளலாம். எப்படியிருந்தாலும் உங்களுக்கு தகுந்த மயக்க மருந்து கொடுத்து அறுவை சிகிச்சை செய்யப்படும். இந்த ஆய்வில் ஏதேனும் பக்க விளைவுகள் ஏற்பட்டால் உங்களுக்கு முழு சிகிச்சை மருத்துவ குழுவினால் அளிக்கப்படும்.

நாள் :

நேயாளியின் கையொப்பம்

/ இடது பெருவிரல் ரேகை

***Ethical
Committee
Approval
Certificate***

CERTIFICATE FOR APPROVAL OF ETHICAL COMMITTEE

To

Dr.K. Sathisha Kumar - PG In Anaesthesia

Dear Dr.K. Sathisha Kumar - PG In Anaesthesia

The Institutional Ethics Committee reviewed and discussed your application for approval of the project entitled

"Comparative study for the purpose of Safety & Efficacy of 0.5% Ropivacaine with adrenaline, 0.5% Ropivacaine with adrenaline, 0.5% Ropivacaine without adrenaline & 0.5% Bupivacaine in Patients Receiving subclavian Perivascular Block."

The following members of the ethics committee were present at the meeting held on 15.04.2010 at the Modernised Seminar Hall, Stanley Medical College, Chennai-1 at 2PM

Dr.R. Nandhini, Director of Pharmacology,

Madras Medical College, Chennai-3 - Chairman of the Ethics Committee

Dr.Usha Sadhasivam, Prof.of Pharmacology,

Stanley Medical College, Chennai - 1- Member Secretary of the Ethics Committee

MEMBERS

Dr.S. Ramasamy

Prof.of Medicine

Dr.S. Deivanayagam

Prof.of Surgery

Dr.Ruckmani

Prof.of O&G

Thiru.A. Senthil Manoharan,

Advocate

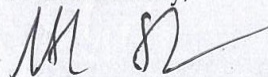
Thiru.P. Mohan,

Social Scientist

We approve the project to be conducted in its presented form.

The Institutional Ethics Committee/Independent Ethics Committee expects to be informed about the progress of the study, any SAE occurring in the course of the study, any changes in the protocol and patient information/informed consent and asks to be provided a copy of the final report.

Yours sincerely,



Member Secretary,
Ethics Committee